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United States
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Office of Research and
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Washington, DC 20460

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July 1993

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EPA

# Urban Soil Lead Abatement Demonstration Project

**Review Draft**
(Do Not Cite or Quote)

## Volume I: Integrated Report

### Notice

This document is a preliminary draft. It has not been formally released by EPA and should not at this stage be construed to represent Agency policy. It is being circulated for comment on its technical accuracy and policy implications.

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# Urban Soil Lead Abatement Demonstration Project

## Volume I: Integrated Report

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Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

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## LIST OF PARTICIPANTS

Urban Soil Lead Abatement Demonstration Projects

Dr. Ann Aschengrau
Boston University School of Public Health
80 East Concord Street, T-355
Boston, MA 02118

Dr. David Bellinger
Children's Hospital
Gardner House Room 455
300 Longwood Avenue
Boston, MA 02115

Dr. Robert Bornschein
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue #56
Cincinnati, OH 45267-0056

Ms. Dawn Boyer
Inorganic Chemistry Department
Lockheed ESC
1050 East Flamingo
Las Vegas, NV 89119

Ms. Merrill Brophy
MDE/Lead & Soil Project
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Richard Brunker
U.S. EPA - Region III
Site Support Section MD-3HW26
841 Chestnut Street
Philadelphia, PA 19107

Mr. Barry Chambers
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Rufus Chaney
U.S. Department of Agriculture
ARC, Building 318 BARC-East
Beltsville, MD 20705

Dr. Julian Chisolm
Kennedy Institute
707 N. Broadway
Baltimore, MD 21205

Dr. Scott Clark
University of Cincinnati
Department of Environmental Health
Mail Stop 56
3223 Eden Avenue
Cincinnati, OH 45267-0056

Ms. Linda Conway-Mundew
University of Cincinnati
Department of Environmental Health
1142 Main Street
Cincinnati, OH 45210

Dr. Robert Elias
U.S. EPA
Environmental Criteria Assessment Office
Mail Drop 52
Research Triangle Park, NC 27711

Dr. Katherine Farrell
Anne Arundell County Health Department
3 Harry S. Truman Parkway
Annapolis, MD 21401

Ms. Beverly Fletcher
U.S. EPA - Region I
Environmental Services Division
60 Westview Street
Lexington, MA 02173

LIST OF PARTICIPANTS (cont'd)

Urban Soil Lead Abatement Demonstration Projects

Mrs. Barbara Gordon
Cincinnati Health Department
3101 Burnet Avenue, Room 309
Cincinnati, OH 45229

Ms. Jo Ann Grote
University of Cincinnati
Department of Environmental Health
1142 Main Street
Cincinnati, OH 45210

Mr. Bill Hanson
Cincinnati Health Department
UC Soil Project
1142 Main Street
Cincinnati, OH 45210

Mr. Reginald Harris
U.S. EPA - Region III
Site Support Section MD-3HW15
841 Chestnut Street
Philadelphia, PA 19107

Mr. Ronald Jones
Cleveland Department of Public Health
1925 East St. Claire Ave.
Cleveland, OH 44114

Dr. Boon Lim
Environmental health Program
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Allan Marcus
Battelle Applied Statistics
100 Park Drive
P.O. Box 13758
Research Triangle Park, NC 27709

Dr. Tom Matte
Public Health Service - Region II
26 Federal Plaza, Room 3337
New York, NY 10278

Ms. Lisa Matthews
U.S. EPA - OS 230
401 M Street, SW
Washington, DC 20460

Mr. J. Todd Scott
The Cadmus Group
3580 Cinderbed Road
Suite 2400
Newington, VA 22122

Mr. Dave McIntyre
U.S. EPA - Region I
Environmental Services Division
60 Westview Street
Lexington, MA 02173

Mr. William Menrath
University of Cincinnati
Department of Environmental Health
1142 Main Street
Cincinnati, OH 45210

Dr. Winkey Pan
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45267-0056

Dr. Dan Paschal
Centers for Disease Control
1600 Clifton Road, NE
Mail Stop F-18
Atlanta, GA 30333

## LIST OF PARTICIPANTS (cont'd)

Urban Soil Lead Abatement Demonstration Projects

Ms. Sandy Roda
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45267-0056

Dr. Charles Rohde
Biostatistics
Johns Hopkins University
615 N. Wolfe Street
Baltimore, MD 21205

Ms. Penny Schmitgen
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45210

Dr. James Simpson
Centers for Disease Control
CEHIC/EHHE, Mail Stop F28
1600 Clifton Road
Atlanta, GA 30333

Dr. Tom Spittler
U.S. EPA - Region I
Environmental Services Division
60 Westview Street
Lexington, MA 02173

Mr. Warren Strauss
MDE/Lead & Soil Project
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Paul A. Succop
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45267-0056

Dr. Pat VanLeeuwen
U.S. EPA - Region V
Technical Support Unit 5HR-11
230 S. Dearborn Street
Chicago, IL 60404

Dr. Harold A. Vincent
U.S. EPA
Quality Assurance Division
Environmental Monitoring Systems
Laboratory - Las Vegas
P.O. Box 93478
Las Vegas, NV 89193-3478

Dr. Michael Weitzman
Chief of Pediatrics
Rochester General Hospital
1425 Portland Avenue
Rochester, NY 14621

LIST OF REVIEWERS

**COPY IS STILL TO COME.**

# LIST OF ABBREVIATIONS AND ACRONYMS

| | |
|---|---|
| AAS | Atomic Absorption Spectroscopy |
| ANCOVA | Analysis of Covariance |
| ANOVA | Analysis of Variance |
| Autoregressive Regression Model | Statistical procedure for multiple linear regression where one variable is regressed on preceding values of the variable as well as other related variables |
| BAL P | Baltimore Study Group with Paint Intervention |
| BAL SP | Baltimore Study Group with Soil and Paint Intervention |
| BOS P | Boston Study Group with Paint Intervention |
| BOS PI | Boston Study Group with Paint and Interior Dust Intervention |
| BOS SPI | Boston Study Group with Soil, Paint, and Interior Dust Intervention |
| CDC | Centers for Disease Control |
| CIN I-SE | Cincinnati Study Group with Interior Dust Intervention, followed by Soil and Exterior Dust Intervention (second year) |
| CIN NT | Cincinnati Study Group with No Treatment |
| CIN SEI | Cincinnati Study Group with Soil, Exterior Dust, and Interior Dust Intervention |
| CORR | Systat Procedure for pair-wise correlations |
| dL | Deciliter. Used here as a measure of blood lead in $\mu$g/dL |
| Double Blind | Analytical audit sample where analyst knows neither that the sample is an audit sample nor the concentration |
| Dust Loading | Mass of dust per unit area |
| ECAO/RTP | Environmental Criteria and Assessment Office/Research Triangle Park |

| | |
|---|---|
| EPA/ORD | EPA/Office of Research and Development |
| EPA/OSWER | EPA/Office of Solid Waste and Emergency Response |
| FAM | Datafile indexed by Family or Living Unit |
| GIS | Geographic Information Systems |
| GLIM | Numerical Algorithms Group software package for a general linear model |
| GLM | SAS procedure for general linear models approximately equivalent to Systat MGLH |
| GSD | Geometric Standard Deviation |
| Hand Dust | Sample taken by wiping the child's hand thoroughly; a measure estimating the ingestion of lead |
| HEPA | High Efficiency Particle Accumulator |
| ICP | Inductively Coupled Plasma Emission Spectroscopy |
| KID | Datafile indexed by child |
| MEANS | SAS Procedure for calculating means |
| MGLH | Systat procedure for general linear models approximately equivalent to SAS GLM |
| NBHD | Datafile indexed by Neighborhood |
| NHANES II | National Health Assessment and Nutrition Examination Survey II |
| NONLIN | Systat program for single response nonlinear regression models |
| P-value | Statistical term for the likelihood that an observed effect differs from zero |
| Pb | Lead |
| Pb Concentration | Mass of Pb per mass of medium (soil, dust, water) |

| | |
|---|---|
| Pb Loading | Mass of Pb per unit area |
| PC | Personal Computer |
| Pearson Correlation Coefficient | Statistical term for one measure of correlation between two variables |
| Project | In this report, "project" refers collectively to the three individual studies that compose the Urban Soil Abatement Demonstration Project. |
| PROP | Datafile indexed by Property |
| QA/QC | Quality Assurance/Quality Control |
| Repeated Measures Analysis | Statistical procedure for analyzing normally distributed responses collected longitudinally |
| RFP | Request for Proposal |
| Round | Period of sampling and data collection during study |
| SARA | Superfund Amendments and Reauthorization Act |
| SAS | Statistical Software Package |
| SES | Socio-economic Status |
| Single Blind | Analytical audit sample where analyst knows sample is an audit sample but doesn't know concentration (see Double Blind) |
| Study | In this report, "study" refers to one of the three individual soil abatement studies that compose the Urban Soil Abatement Demonstration Project. |
| SYSTAT | Statistical Software Package |
| U.S. EPA | U.S. Environmental Protection Agency |
| USLADP | Urban Soil Lead Abatement Demonstration Project |
| XRF | X-Ray Fluorescence |

| | |
|---|---|
| XRFE | Exterior measurements of Lead-based paint using portable XRF instruments |
| XRFI | Interior measurements of Lead-based paint using portable XRF instruments |

# 1. EXECUTIVE SUMMARY

## 1.1 SCOPE OF REPORT

This document describes the results of the Urban Soil Lead Abatement Demonstration Project (USLADP) from the perspective of the reanalysis of data from the three studies that participated in the project. Taken individually, the reports of the three studies could support three different conclusions concerning the feasibility of reducing lead exposure by abating soil. Collectively, a common picture emerges that places a significant role for soil abatement in the total scheme of lead exposure reduction. Because of the common design of the three studies and their focus on key experimental parameters, it is possible to treat certain key parameters as an integrated data set and analyze the results of each study separately but identically. This report presents the results and conclusions drawn from a detailed statistical reanalysis of the integrated data set.

The Urban Soil Lead Abatement Demonstration Project, known also as the Three City Study, was authorized in 1986 under Section 111(b)(6) of the Superfund Amendments and Reauthorization Act (SARA). The purpose of the project was to determine whether abatement of lead in soil could reduce the lead in blood of inner city children.

Until 1986, the concept of soil abatement had been applied mainly to residential (usually nonurban) areas located near Superfund sites where lead was a major contaminant. The decision to abate soil was usually based in part on the distribution of blood lead concentrations within the population of children. There were few, if any, attempts to measure the effects of this abatement and little or no opportunity to follow up with further studies of the results.

This project is three coordinated longitudinal studies of urban children where intervention into the pathway of lead exposure was expected to reduce the children's blood lead. There have been many cross-sectional studies of childhood lead exposure under a wide range of exposure conditions. These studies showed that differences in lead exposure produced differences in blood lead concentrations. They did not show that changes in exposure produce changes in blood lead. This would require a longitudinal study. Before now, there were few longitudinal studies; none involved extensive intervention, and some showed that increasing lead exposure results in increasing blood lead concentrations. The unique aspect of this project is that it measures a response to intervention, not contamination.

There is a difference between children who are suddenly placed into a "clean" environment and those who have lived continuously in a clean environment. Because of the physiology of lead mobilization in body tissues, there is a difference between the rate of change in a population with increasing lead exposure and in one with decreasing exposure. In other words, the decrease in blood lead concentrations in response to intervention was not expected to be at the same rate as the increase in blood lead concentrations with increasing exposure.

The project began in December 1986 with the appointment of a Steering Committee to develop recommendations for implementing the SARA lead-in-soil demonstration project. A panel of experts was formed in March 1987 to set the criteria for selection of sites and the minimum requirements for a study at each site. An early decision was that the options for soil abatement methods were limited, because only excavation and removal had been used in similar programs. Therefore, there would be no attempt in the project to evaluate alternative methods of abatement because of limited time and resources. The panel met again in April 1987 to discuss technical issues and study designs and evaluate technical criteria for selection of urban areas as potential soil-lead abatement demonstration project sites. They established site selection criteria that in December, 1987 led to the selection of Boston, Baltimore, and Cincinnati as the participating sites.

## 1.2 BACKGROUND AND OVERVIEW

In the mid 1980s, concern for childhood lead exposure increased with mounting evidence that urban environments were exposed to lead in soil to a degree that might be related to potential health effects. Evidence for this concern came from the apparent correlation between the incidence of high blood lead concentrations and high concentrations of lead in residential soils. At that time, there were several other sources of exposure that could potentially account for unusually high blood lead in a population of urban children. Among these were lead in the air (primarily from automobile emissions), lead in food (primarily from canned foods with lead soldered side seams), lead in drinking water (primarily from lead pipes or newly soldered copper pipes), and lead in paint. The lead in the soil was believed to be a mixture of lead from the atmosphere and lead from exterior paint. At that time, regulations were in place that would remove nearly all tetraethyl lead from gasoline by the end of 1986, and there was a voluntary program among food processors to phase out cans with lead soldered side seams and use only cans without lead solder. The relationship between soil lead and blood lead is an indirect relationship in the sense that children most commonly do not eat soil directly but ingest small amounts of dust derived, in part, from this soil. In the child's environment, soil is only one of several sources of lead. Likewise, the lead in blood reflects not only exposure from these sources but also the biokinetic processes that distribute and redistribute lead between blood and other body tissues, especially bone tissue.

### 1.2.1 Study Designs

Each study was designed around the concept of participating families within a definable neighborhood. There were two or three study groups in each study, with one to three neighborhoods in a study group. Each study group was evaluated during three phases: preabatement, abatement and postabatement. This means that prior to and after abatement, the environment of the child was extensively evaluated through measurements of lead in soil, dust, drinking water, and paint, and through questionnaires about activity patterns, eating habits, family activities, and socioeconomic status. The objective of the preabatement phase was to determine the exposure history and status (stability of the blood lead and environmental measures) prior to abatement. During the abatement phase, all possible measures were taken to prevent exposure that might result from the abatement activities. During the postabatement phase, samples were taken to measure the duration of the effect of soil abatement and to detect possible recontamination.

Because of the complex nature of this exposure assessment, intermediate exposure indices, such as street dust, house dust, and hand dust were measured wherever possible. This required the development of new sampling and analysis protocols that were not generally available in the scientific literature. These protocols were developed through a Scientific Coordinating Committee composed of representatives from each study, the three Regional offices, the CDC, EPA/Office of Solid Waste and Emergency Response, EPA/Office of Research and Development, and Battelle/Research Triangle Park, NC. They were largely complete by the start of the preabatement phase in January 1989.

Table 1-1 describes the study groups and the form of intervention. The Cincinnati study design used intervention on the neighborhood scale, where the soil in parks, play areas and other common grounds were abated, and paved surfaces in the neighborhood were cleaned of exterior dust lead. In Boston and Baltimore, only the soil on the individual properties was abated. Table 1-2 describes the study design characteristics for each of the three studies and their respective participant groups. The general characteristics are that soil lead concentrations are typically high in Boston, and it is common to find lead in drinking water and in both exterior and interior paint. In the Boston areas studied, housing is typically single family with relatively large lot sizes. In the Baltimore neighborhoods, nearly every house had lead-based paint, the houses were mixed single and multifamily, and the lots were smaller than Boston lots, with typical yards less than 100 square meters. Residential units in Cincinnati were mostly multifamily with little or no soil on the residential parcel of land.

Figure 1-1 illustrates the generalized concept of human exposure to lead, showing the pathways of lead from the several sources in the human environment to four compartments immediately proximal to the individual. One of these proximal sources, dust, is the route of concern in this project. Figure 1-2 expands this dust route to show the complexity of the many routes of dust exposure for the typical child. The strategies for intervention used in this project were designed to interrupt the movement of lead along one or more of these pathways.

Intervention is defined here as the interruption of the flow of lead along an exposure pathway. There were three forms of intervention in this project: soil abatement, dust removal, and paint stabilization. Soil abatement was by excavation and removal. If done correctly, this abatement should establish an effective and persistent barrier to the dust movement. Dust intervention was by vacuuming, wet mopping, and, in some cases, replacement of rugs and upholstered furniture. Cincinnati and Boston performed interior dust abatement, and Cincinnati removed neighborhood dust with mechanical sweepers and hand tools. Dust intervention was not expected to be permanent, because dust continually flows through the human environment. Instead, the removal of dust with elevated lead

TABLE 1-1. DESCRIPTION OF STUDY GROUPS AND TYPES OF INTERVENTION

| Treatment Group Name[a] | Cross-Reference to Individual Study Report | Description of Treatment |
|---|---|---|
| | | BOSTON |
| BOS SPI | Study Group | Soil and interior dust abatement, and exterior and interior paint stabilization at beginning of first year, no further treatment |
| BOS PI | Control Group A | Interior dust abatement and exterior and interior paint stabilization at beginning of first year |
| BOS P | Control Group B | Exterior and interior paint stabilization at beginning of first year |
| | | BALTIMORE |
| BAL SP | Study Area | Soil abatement and exterior paint stabilization at beginning of first year, no further treatment |
| BAL P-1[b] | Control Area | Exterior paint stabilization at beginning of first year, no further treatment |
| BAL P-2[b] | Study Area | Exterior paint stabilization at beginning of first year, no further treatment |
| | | CINCINNATI |
| CIN SEI | Area A | Soil, exterior dust, and interior dust abatement at beginning of first year, no further treatment |
| CIN I-SE-1[c] | Area B, Back Street and Findlay neighborhoods | Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment |
| CIN I-SE-2[c] | Area B, Dandridge neighborhood | Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment |
| CIN NT | Area C | No treatment, soil abatement at end of study |

[a]The treatment group designation indicates the location of the study (BOS = Boston, BAL = Baltimore, CIN = Cincinnati), the type of treatment (S = soil abatement, E = exterior dust abatement, I = interior dust abatement, P = loose paint stabilization, NT = no treatment).
[b]Treated as one group in the Baltimore report, analyzed separately in this report.
[c]Treated as one group in the Cincinnati report, analyzed separately in this report.

## TABLE 1-2. NUMBER OF PROJECT PARTICIPANTS BY ROUND[a]

| Study | | | | | | |
|---|---|---|---|---|---|---|
| BOSTON | Round 1 | Round 3 | Round 4 | | | |
| Middate | 10/17/89 | 4/9/90 | 9/12/90 | | | |
| Children[b] | 150 | 146 | 147 | | | |
| Famlies[c] | 125 | 121 | 122 | | | |
| Properties[d] | 100 | 96 | 97 | | | |
| BALTIMORE | Round 1 | Round 2 | Round 3 | Round 4 | Round 5 | Round 6 |
| Middate | 10/25/88 | 4/1/89 | 2/17/90 | 1/27/91 | 6/7/91 | 9/3/91 |
| Children[b] | 408 | 322 | 269 | 200 | 196 | 187 |
| Families[c] | 200 | 203 | 157 | 116 | 110 | 109 |
| Properties[d] | 193 | 201 | 156 | 114 | 108 | 108 |
| CINCINNATI | Round 1 | Round 3 | Round 5 | Round 7 | Round 9 | |
| Middate | 7/6/89 | 11/14/89 | 7/1/90 | 11/17/90 | 6/16/91 | |
| Children[b] | 201 | 185 | 219 | 198 | 169 | |
| Families[c] | 71 | 67 | 66 | 94 | 82 | |
| Properties[d] | 141 | 129 | 124 | 124 | 124 | |

[a]Number shown is based on samples taken and does not include individuals enrolled but not sampled.
[b]Based on number of children sampled for blood.
[c]Based on number of households sampled for dust.
[d]Based on number of properties (Boston, Baltimore) or soil parcels (Cincinnati) sampled.

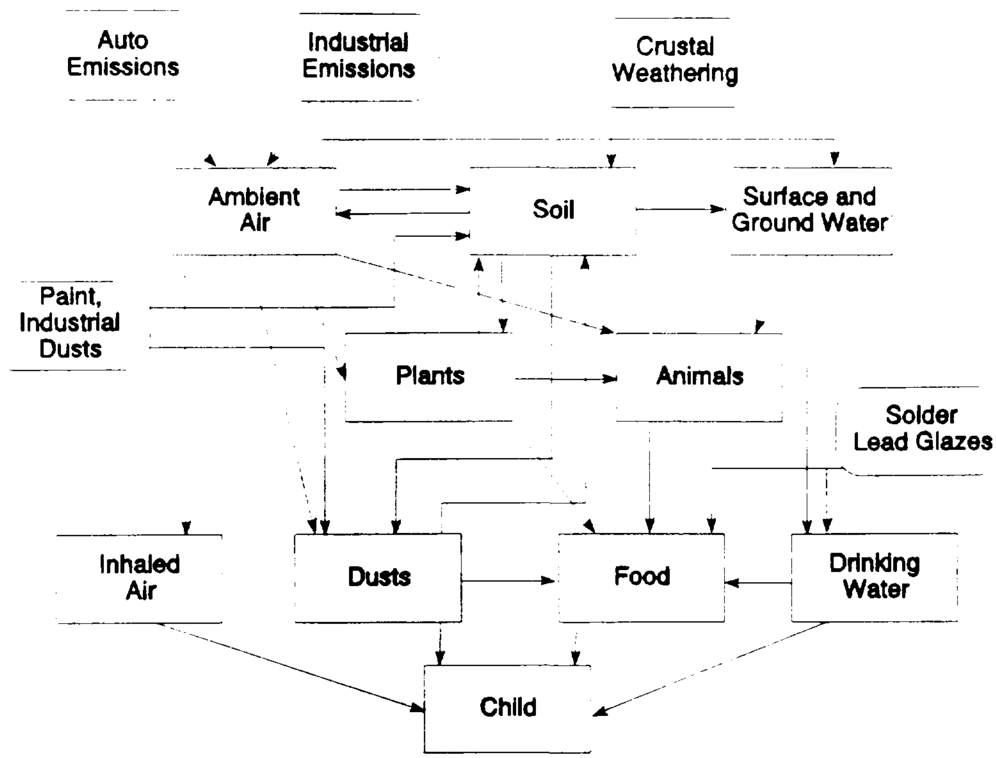


**Figure 1-1. Generalized concept of the sources and pathways of lead exposure in humans.**

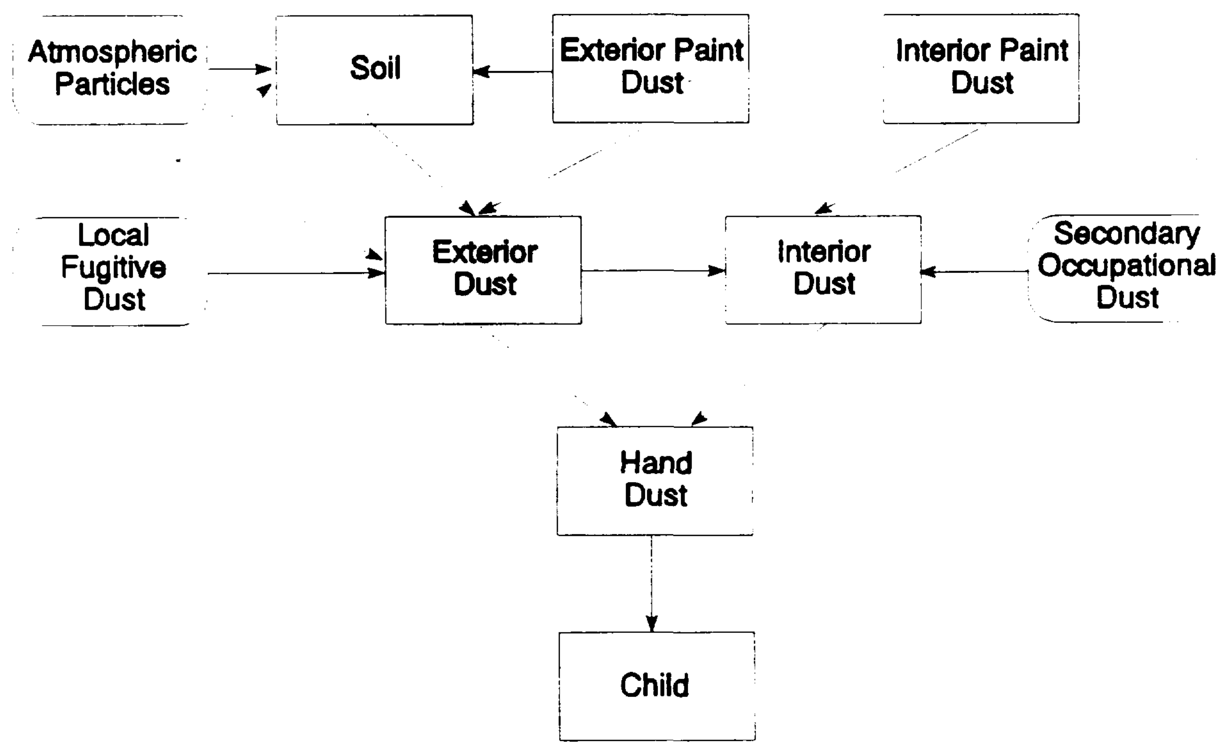


**Figure 1-2. Typical pathways of childhood exposure to lead in dust.**

concentrations was expected to enhance the impact of soil abatement on the child's environment.

In the home, dust is a mixture of exterior dust and soil, interior weathering products including paint flakes and chips, workplace dust carried home by adults, and dust generated from human activities within the household. It is believed that most of the mass of the interior dust originates from soil immediately exterior to the home, but this can vary greatly by the types of family activities. Nevertheless, in the absence of lead-based paint inside the home, it would seem reasonable to assume that most of the lead in household dust comes from soil.

Many of the Boston and Baltimore households selected for the project had chipping and peeling lead-based paint, both interior and exterior. In order to reduce the impact of this paint, much of which was lead-based paint, the walls and other surfaces were scraped and smoothed, and repainted to reduce the impact of lead-based paint on the pathways of lead exposure. It is important to note that this approach in not a full scale paint abatement and was not designed to place a permanent barrier between the paint and the child. Paint stabilization was used on exterior and interior surfaces in Boston, and on exterior surfaces in Baltimore. Paint stabilization was not used in Cincinnati because the lead-based paint had been removed from these homes in the early 1970s.

The frequency and timing of sampling relative to abatement and seasonal cycles is an important aspect of this project. The original design focussed on sampling blood lead during the late summer, as it was known that the seasonal cycle is highest at that time. Where this schedule could not be adhered to, an effort was made to schedule the followup blood lead sampling to characterize this cycle and possibly permit extrapolation to the summer peaks.

In order to accurately measure the effectiveness and persistency achieved by soil abatement, and the impact of this abatement on reducing lead exposure for children, the sampling and analysis plans for soil and dust required robust quality control and quality assurance objectives. Protocols were developed to (1) define sampling schemes that characterize the expected exposure to soil for children; (2) collect, transfer, and store samples without contamination; and (3) analyze soil, dust, handwipe, and blood samples in a manner that would maximize interlaboratory comparison.

## 1.3 BRIEF DESCRIPTION OF HOW INTERVENTION WAS PERFORMED

A total of 93 Boston properties were abated. The information on area treated and volume of soil removed from these properties appears in Table 1-3.

TABLE 1-3. SOIL ABATEMENT STATISTICS FOR THE THREE STUDIES

| | Boston | Baltimore | Cincinnati |
|---|---|---|---|
| Number of properties[a] | 36 | 56 | 171 |
| Surface area ($m^2$) | 7,198 | 4,100[b] | 12,089 |
| Volume soil removed ($m^3$) | 1,212 | 690 | 1,813 |
| Surface area/property ($m^2$) | 200 | 73 | 71 |
| Volume soil/property ($m^3$) | 34 | 11[b] | 11 |

[a]Includes only properties abated during the study. Properties abated at the end of the study, where no further sampling was reported, are not included in this analysis, but are included in the individual study reports. In Cincinnati, a property is the location of the soil abatement, not the location of the child's residence.
[b]Surface area not provided by Baltimore report. Calculated using Boston vol/surf ratio, which is equivalent to an average removal depth of 17 cm.

In Baltimore, 63 properties in the BAL SP treatment group (see Table 1-1) were abated between August and November 1990. An additional seven properties that did not meet the requirements for abatement were transferred to the control group (BAL P). Unpaved surfaces were divided into areas on each property, usually front, back, and one side; any area with soil lead concentrations above 500 $\mu g/g$ was abated entirely.

Within each neighborhood, the Cincinnati study identified all sites with soil cover as discrete study sites. The decision to abate was based on soil lead concentrations for each parcel of land, and for the depth to which the lead had penetrated. Lead was measured in the top 2 cm, and at a depth of 13 to 15 cm. If the concentration in the top sample was greater than 500 $\mu g/g$, the soil was abated. Additional parcels were abated if there was evidence that lead had penetrated into the soil profile.

Exterior dust abatement was performed in the Cincinnati study only. The approach to this abatement was to identify all types of paved surfaces where dust might collect, obtain permission to sample and abate these areas and to clean them once with vacuum equipment, suitable for the area, that had previously been tested and shown to remove about 95% of the available dust on the area. The groups of surfaces selected were streets, alleys, sidewalks, parking lots, steps, and porches. For data analysis, these were grouped as (1) targeted (steps and porches); (2) streets, sidewalks, and alleys; and (3) parking lots and other locations.

The exterior dust measurements in the Cincinnati study (and the interior dust measurements of all three studies) were made in a manner that determined the lead concentration ($\mu$g Pb/g dust), the dust loading (mg dust/$m^2$), and the lead loading ($\mu$g Pb/$m^2$) for the surface measured. This required that a dry vacuum sample be taken over a prescribed area, usually 0.25 to 0.5 $m^2$. It is important to note that dust abatement is not expected to cause an immediate change in the lead concentration on dust surfaces, only in the dust and lead loading.

Household dust was abated in the Boston and Cincinnati studies, but not in Baltimore. The BOS SPI and CIN SEI groups (see Table 1-1) received interior dust abatement at the same time as soil abatement, the BOS PI group received interior dust abatement without soil abatement, and the CIN I-SE-1 and CIN I-SE-2 groups received interior dust abatement in the first year, followed by soil and exterior dust abatement in the second year.

In Boston, interior dust abatement was performed after loose paint stabilization. Families were moved off site during interior dust abatement. Hard surfaces (floors, woodwork, window wells, and some furniture) were vacuumed, as were soft surfaces such as rugs and upholstered furniture. Hard surfaces were also wiped following vacuuming. Common entries and stairways outside the apartment were not abated.

The Cincinnati group performed interior dust abatement after exterior dust abatement and also moved the families off site during this activity. Vacuuming, was followed by wet wiping with a detergent. They vacuumed hard surfaces and replaced one to three carpets and two items of upholstered furniture per housing unit. Their previous studies had shown that these soft items could not be cleaned effectively with vacuuming alone.

Most homes in the Cincinnati group had received paint abatement 20 years prior to the project, but in Boston and Baltimore lead-based paint occurred in nearly every home. Because full paint abatement was not within the scope of this project, the alternative was to retard the rate of movement of paint from the walls to household dust to the extent possible. The interior and exterior surfaces of all Boston homes and the exterior surfaces of all

Baltimore homes received loose paint stabilization approximately one week before soil abatement.

In Boston, loose paint stabilization consisted of removing chipping and peeling paint and washing the surfaces. Window wells were painted with a fresh coat of primer. Baltimore homes were wet scraped over the chipping and peeling surfaces, followed by vacuuming. The entire surface was primed and painted with two coats of latex paint.

## 1.4 BRIEF SUMMARY OF INDIVIDUAL STUDY REPORTS

### 1.4.1 Summary of the Boston Study

The Boston study retained 149 of the original 152 children enrolled, although 22 children moved to a new location but were retained in the study. Children with blood lead concentrations below 7 μg/dL or above 24 μg/dL had been excluded from the study and two children were dropped from the data analysis when they developed lead poisoning, probably due to exposure to lead-based paint at another location.

Baseline characteristics (age, SES, Soil lead, dust lead, drinking water lead, and paint lead) were similar for the three study groups (BOS P, BOS SP, BOS SPI). The pre-abatement blood lead concentration was higher for BOS P. The proportion of Hispanics was higher in BOS P the BOS SP or BOS SPI, and the proportion of Blacks was lower. There was a larger proportion of male children in BOS P.

Data were analyzed by analysis of covariance (ANCOVA), which showed a significant effect of group assignment (intervention) for both the BOS SP and BOS SPI groups. These results did not change with age, sex, socioeconomic status, or any other variable except race and paint. When the paint variable was added, the effect was diminished; when the race variable was added, the effect became insignificant.

Although designed and conducted to produce rigorous results, the study has several limitations. Participants were chosen to be representative of the population of urban preschool children who are at risk of lead exposure by using the Boston Childhood Lead Poisoning Prevention Program to identify potential participants from neighborhoods with the highest rates of lead poisoning and by using as wide a range of blood lead levels as was practical. Since no study subjects had blood lead levels below 7 μg/dL or in excess of

24 $\mu$g/dL at baseline, the study provides no information about the effect of lead contaminated soil abatement for children with these lead levels. Similarly, a different effect might have been found for children who had a greater blood lead contribution from soil, such as in communities with smelters or other stationary sources where soil lead levels are substantially higher than those seen in this study, or where differences in particle size result in differences in bioavailability.

It is possible that the intervention would have been associated with a greater reduction in children's blood lead levels had they been followed for a longer period of time. In addition, all children in the study were exposed to lead contaminated soil prior to enrollment and so we are unable to investigate whether exposure to lead contaminated soil in the first year of life is associated with higher blood lead levels. Lastly, the unit of abatement was the single premises rather than clusters of premises. It is possible that the effect of lead contaminated soil abatement on children's blood lead levels would have been greater had we also removed lead contaminated soil from properties that surrounded Study Group children's premises.

In conclusion, this intervention study suggests that an average 1,856 ppm reduction in soil lead levels results in a 0.8 to 1.6 $\mu$g/dL reduction in the blood lead levels of urban children with multiple potential sources of exposure to lead.

This study provides information about soil abatement as a secondary prevention strategy, that is the benefit to children already exposed to lead derived, in part, from contaminated soil. It can not be used to estimate the primary prevention effect of soil abatement. Since children's postabatement blood lead levels reflect both recent exposure and body burdens from past exposure, the benefit observed is probably less than the primary prevention benefit, that is the benefit of abating lead contaminated soil before children are exposed to it so as to prevent increases in blood levels and body stores.

### 1.4.2 Summary of the Baltimore Study

The Baltimore study recruited 472 children, of whom 185 completed the study. Of those that completed the study, none were excluded from analysis. The recruited children were from two neighborhoods, originally intended to be a study and a control group. Because soil concentrations were lower than expected, some properties in the study group did not receive soil abatement. The Baltimore report transferred these properties to the control group. In this report, the low soil properties in the study group are treater as a separate group.

Because of logistical problems, there was an extended delay between recruitment and soil abatement that accounted for most of the loss of the participating families from the project. In their report, the Baltimore group applied several statistical models to the two populations to evaluate the potential bias from loss of participating children. These analyses showed the two populations remained virtually identical in demographic, biological and environmental properties.

The Baltimore study was not designed to focus on measurements of the movement of lead through the child's environment. Repeat measurements of soil were on abated properties only, to confirm abatement. There were no measurements of exterior dust, no interior paint stabilization, and no followup measurements of house dust. Rather, the study design focused on changes in biological parameters, hand dust and blood lead over an extended period of time.

Including the pre-study screening measurements of hand dust and blood lead in the original cohort of participants, the Baltimore study made six rounds of biological measurements that spanned twenty months. It is unusual to have a data set of this composition and quality. In this integrated report, the baltimore blood lead measurements were the basis for determining the key parameters in the seasonal cycle conversion factor equation discussed in Section 3.3.5.1.

Soil was abated between the third and fourth rounds of biological measurements. The mean soil decrease was 550 $\mu$g/g. At Round 4, the blood lead concentrations were about 0.5 $\mu$g/dL lower in the study group than in the control, or 1 $\mu$g/dL per decrease of 1,000 $\mu$g/g in soil, which is comparable to the response observed in the Boston study. By Rounds 5 and 6, the study group blood lead concentrations had returned to their preabatement levels and were in fact higher than the control group.

From the perspective of the Baltimore study alone, it is reasonable to conclude, as the Baltimore report did, that soil abatement has no effect on children's blood lead. But from the perspective of the Boston study, where a blood lead reduction of the same magnitude was found to be persistent when house dust abatement was performed, and from the perspective of the Cincinnati study, where blood lead concentrations were shown to rise and fall in tandem with house dust concentrations, the results of the Baltimore study are consistent with the observation that soil abatement, in conjunction with other environmental interventions, can permanently reduce exposure to lead.

### 1.4.3 Summary of the Cincinnati Study

The Cincinnati study recruited 307 children, including 16 children born to participating families during the study, and an additional 50 children who were recruited after the beginning of the study. In their final report, the Cincinnati group excluded these children who were recruited after the start of the study, plus 31 children who were living in nonrehabilitated housing suspected of having lead-based paint, and four children (in two families) who had become lead-poisoned from other causes. Thus, data for 210 children were analyzed in the Cincinnati report and these same children were included in this integrated report.

The Cincinnati study achieved effective and persistent abatement of soil on the 140 parcels of land scattered throughout the neighborhoods. In CIN SEI, where soil abatement was performed in the first year, the arithmetic mean concentration dropped from 680 $\mu$g/g down to 134 $\mu$g/g. In the two groups where soil abatement occurred in the second year, CIN I-SE-1 and CIN I-SE-2, the soil lead concentration dropped from 262 $\mu$g/g to 125 $\mu$g/g and 724 $\mu$g/g to 233 $\mu$g/g, respectively.

If soil were the only source of lead in the neighborhoods, exterior and interior dust should have responded to the reduction in soil lead concentrations. Exterior dust lead loading decreased following both soil and dust abatement, but returned to preabatement levels within one year. In their report, the Cincinnati group concluded that recontamination of exterior dust began soon after abatement. They observed corresponding changes in house dust, hand lead, and blood lead that paralleled changes in exterior dust. Because blood lead concentrations also decreased in the control area, the Cincinnati group concluded that there is no evidence for the impact of soil and dust abatement on blood lead concentrations. This integrated report concludes, through a more detailed structural equation analysis, that there is a strong relationship between exterior dust and interior dust in this subset of the Cincinnati study where the impact of lead-based paint was minimized. From the perspective of all three studies, this means that when neighborhood and living unit sources of lead are removed, exposure is reduced.

The Scientific Coordinating Committee attempted to establish uniformity among the three studies for several aspects of the project. Although there were differences in form and content, each study plan contained:

1. a statement of the objectives of the study;

2. a testable hypothesis that provided direction and focus to the study;

3. protocols for collecting and analyzing the data;

4. an array of treatment groups that addressed all features of the hypothesis;

5. measures to be taken to ensure that all phases of the study would be conducted as planned; and

6. procedures by which the results of the study would be processed, analyzed, and interpreted.

The objectives, protocols for sampling and analysis, QA/QC plans, and data processing procedures were nearly identical for all three studies. Elements that differed slightly among the three studies were the hypotheses and the array of treatment groups. The hypotheses differed only slightly, as seen from the following statements.

The central hypothesis of the Urban Soil Lead Abatement Demonstration Project is:

> *A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*

The formal statement of the Boston hypothesis is:

> *A significant reduction (equal to or greater than 1,000 μg/g) of lead in soil accessible to children will result in a mean decrease of at least 3 μg/dL in the blood lead levels of children living in areas with multiple possible sources of lead exposure and a high incidence of lead poisoning.*

The Baltimore hypothesis, stated in the null form, is:

> *A significant reduction of lead (≥ 1,000 μg/g) in residential soil accessible to children will not result in a significant decrease (3 to 6 μg/dL) in their blood lead levels.*

The Cincinnati hypothesis was separated into two parts:

(1) *A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*

(2) *Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in blood lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

Secondary hypotheses in the Cincinnati study are:

(3) *A reduction of lead in residential soil accessible to children will result in a decrease in their hand lead levels.*

(4) *Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in hand lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

The array of treatment groups differed considerably among the three studies (Table 1-1). Each treatment group, however, had several features in common. All groups were taken from one to three demographically similar neighborhoods. All groups had some prior evidence of elevated lead exposure, usually a greater than average number of reports of lead poisoning. Each group received the same pattern of treatment: baseline phase for 3 to 18 mo, intervention (except for controls), and followup for 12 to 24 mo.

In each treatment group, even the controls, there was an attempt to minimize the impact of lead-based paint. In Boston, this was done by paint stabilization of both interior and exterior paint. In Baltimore, only exterior paint was stabilized. Therefore, in these two studies, the effects of soil abatement should be evaluated in the context of some intervention for lead-based paint. In Cincinnati, most of the living units had been abated of lead-based paint more than 20 years before the start of the study. Those that had not been abated were measured but not treated prior to the study and were in included in the final analysis.

Another difference between the studies was the parallel intervention scheme used in Boston and Baltimore, compared to the staggered scheme used in Cincinnati. In other words, intervention in Boston (and Baltimore) took place at the same time for all treatment groups, and the followup period was of the same duration. But in Cincinnati, the intervention was delayed for one group, CIN I-SE, such that followup varied between 12 and 24 mo.

## 1.5 SUMMARY OF RESULTS AND STATISTICAL INFERENCES

From the perspective of the child's environment, changes in the soil lead concentration are expected to bring about changes in the house dust concentration, the hand dust, and the blood lead concentration. In each of the three studies, the soil lead concentrations were reduced to approximately 50 $\mu$g/g in the study area, and for most children, there was a measurable reduction of blood lead, although not always statistically significant. When corrected for seasonal and age related cyclic variations on blood lead, the impact was even greater, and the effect was maximized when street dust and house dust were also removed from the environment.

### 1.5.1 Quality of the Data

In the absence of certified standards for soil and dust, it was necessary to put into place a program that would ensure that analyses performed by the three participating laboratories would be internally accurate and externally consistent with similar analyses by other researchers. This program consisted of identifying acceptable analytical and instrumental methods, establishing a set of soil and dust standards, and monitoring the performance of the participating laboratories through an external audit program.

Because chemical extraction of 75,000 soil and dust samples presented a costly burden on the project both in terms of time and expense, and because of the advantage of nondestructive analysis for a project of this nature, the Scientific Coordinating Panel recommended the use of XRF for soil analysis on the condition that a suitable set of common standards could be prepared for a broader concentration range and that a rigorous audit program be established to ensure continued analytical accuracy. Two groups, Boston and Baltimore, elected to use XRF for dust analysis also, whereas Cincinnati opted for hot nitric extraction with AAS. During the study, the Baltimore group recognized problems with analyzing dust by XRF when the sample size was small, less than 100 mg. They reanalyzed the dust samples by AAS and reported both measurements. In Boston, this problem was solved by compositing the floor dust samples for XRF analysis, reporting one floor dust sample per housing unit.

During the project, there were two rounds of soil and dust intercalibration measurements, one near the beginning and one at the completion of the soil and dust analyses. These exercises involved the three participating laboratories and two additional laboratories for each exercise. These exercises provided the basis for the conversion factors used to compare soil and dust data between laboratories, and for the evaluation of the performance of each laboratory in the audit sample program.

For soil and dust, multiple measurements must be reduced to a single representative data point for each property or living unit for each round of measurement. This measure of central tendency was reported differently for each of the three studies. Boston used the arithmetic mean, giving equal weight to all values. Cincinnati used the geometric mean, which gives lesser weight to the extremes and is always lower than the arithmetic mean except when the distribution is perfectly normal. Baltimore used a tri-mean approach that gives lesser weight to the extremes while not underestimating exposure for right-skewed distributions.

Each study maintained rigorous standards for database quality. These included double entry, 100% visual confirmation, and standard procedures for detecting outliers. Additional errors were found during the preparation of this report and corrected prior to use in this report. None of these errors would have impacted the conclusions drawn by the individual study. To minimize further errors that might impact this report, statistical procedures were repeated to replicate the results of each report and confirm the exactness of data.

### 1.5.2 Effectiveness and Persistency of Intervention

Soil abatement was found to be effective in all three studies and persistent in both Boston and Cincinnati. There was no measure of soil abatement persistency in Baltimore. Evidence for exterior dust recontamination in Cincinnati suggests lack of effectiveness or persistency of abatement. Further analysis of the data may resolve the issue of the source of this recontamination.

Interior dust abatement was effective and persistent in both Boston and Cincinnati, even though some recontamination occurred in Cincinnati in response to the exterior dust recontamination. Paint stabilization appeared to have some impact on exposure, but there were no measures of effectiveness or persistency.

### 1.5.3 Summary of Statistical Inferences

From the Boston study, a statistical analysis of the data shows that when dust lead and soil lead levels show a persistent decrease as a result of effective abatement, blood lead levels also show a persistent decline. The postabatement blood lead levels are lower when postabatement dust lead levels are persistently lower over a long time.

From the Baltimore study, the analyses show that soil lead abatement had little effect on the primary factors responsible for elevating child blood lead levels, which appear to be interior lead-based paint and interior dust lead.

In Cincinnati, there appear to be additional sources of environmental lead exposure that had different effects on the neighborhoods during the course of the study and were not related to the abatement methods used in the Cincinnati study.

The analysis of the data from the three studies showed evidence that blood lead responds to changes in environmental lead. This suggests that abatement of any type and to any degree will cause a reduction in the blood lead of children. All three studies and all groups within each study produced data supporting this conclusion, although not statistically significant in two of the groups in the Baltimore study.

All three studies also showed evidence for a quantifiable impact of intervention. This may have been intervention from soil abatement, dust abatement, or paint stabilization. In Baltimore, this impact was temporary at best and was marginally significant. In Cincinnati, the impact was quickly swamped by other sources of environmental lead. In Boston, the impact was persistent. The best estimate for this effect is 1 $\mu$g/dL per 1,000 $\mu$g/g decrease in soil. Similar decreases in exterior dust would be expected to have a similar effect.

There is evidence from all three studies that lead moves throughout the child's environment. This means that lead in soil becomes lead in street or playground dust, lead in paint becomes lead in soil, and lead in street dust becomes lead in house dust. A more detailed analysis of the data may show the relative contribution from two or more sources, but the present analyses confirm that this transfer takes place. In the Baltimore study, there was statistical evidence for implied causal pathways, such as paint to exterior dust, but in the Boston and Cincinnati studies, the pathways were explicit.

Finally, there is evidence for the continued impact of nonabated sources following abatement. This means that abatement of soil probably does not reduce the contribution of paint lead to the child's exposure.

## 1.6 SUMMARY STATEMENT OF PROJECT CONCLUSIONS AND THEIR IMPLICATIONS

### 1.6.1 Project Conclusions

This report concurs with the results reported by the individual studies. The reanalysis of the individual study data sets, where performed in the same manner as the report, revealed no evident errors in statistical analysis. The statistical analyses revealed that the relationship between environmental lead and blood lead was more or less uniform across all three studies. When the environmental lead increased, the blood lead increased, and when environmental lead decreased, blood lead decreased.

The results of these three studies demonstrated a clear relationship between environmental lead and blood lead. There were few instances where changes in the blood lead concentrations could not be attributed to changes in environmental lead. Unexplained changes appeared to cause an increase in blood lead concentrations. The fact that both hand dust loads and blood lead concentrations responded accordingly gives credence to the strong link between environmental lead and blood lead.

Finally, the project results shed additional light on the well-known phenomenon of seasonal cycles in blood lead concentrations. The rare opportunity to evaluate three independent longitudinal studies with similar sampling and analysis protocols led to the conclusion that the amplitude of the cycle is roughly 15% in all three cities, that the peak occurs about August 15-20, and that these factors appear to be independent of environmental lead.

In terms of changes attributed to intervention, all three studies observed a quantifiable change in response to intervention. The analyses in Chapter 4 show that, although not always statistically significant, this quantifiable response to intervention is consistent even at low levels of environmental lead. Normalized to a decrease in soil of 1,000 $\mu$g/g, the response appears to be in the range of 0.7 to 1.5 $\mu$g/dL. This suggests that there is no plateau, within the ranges measured in this project, where the removal of environmental lead will not produce a corresponding reduction in blood lead concentrations.

It is expected that the data of this project will be analyzed and reanalyzed by many qualified scientists to extract further information from this massive database. As the need arises and time permits, further analyses may shed light, through additional structural equation modeling and meta analysis, on the intricate pattern of environmental lead exposure in urban neighborhoods. Much of the data are amenable to Geographic Information System (GIS) studies and can be useful to state and local public health officials in assessing the extent of lead exposure in their own domain. The efforts of the many investigators in this project have produced much useful information and, as usual in a well-planned and executed study, raised many additional questions.

### 1.6.2 Implications

In spite of the recent successes in reducing exposure to lead by removing lead from gasoline and canned food, lead exposure remains a complex issue. This integrated report attempts to assess exposure to lead in soil and house dust. It is only one component of the risk assessment process and cannot by itself be the sole basis for a risk management decision. However, the observations and conclusions are based on sound scientific measurements and reasonable interpretations of these measurements. A thorough understanding of the results of this project can provide guidance for regulatory decisions and public health policies.

This report concludes that a reduction in environmental lead corresponding to a decrease of 1,000 $\mu$g/g in soil will result in a reduction of about 1 $\mu$g/dL in blood lead. Although this modest decrease suggests that soil abatement as a form of environmental intervention would not be particularly effective in clinical treatment of a lead poisoned child, in an environmental intervention program where the goal is to reduce the incidence of blood lead concentrations above 10 $\mu$g/dL, this small change could reduce this incidence by 10 to 15%.

Lead in soil and lead-based paint are closely linked in the child's environment. If there is exterior lead-based paint, then soil lead is likely to be elevated. If there is interior lead-based paint, then soil abatement measures to reduce the impact of soil lead on house dust will be ineffective. Public health programs designed to reduce lead exposure will not achieve that objective unless both paint and soil abatement are implemented.

From a regulatory standpoint, where the goal is to determine a safe level of lead in soil, this report concludes that abatement of soil above 500 $\mu g/g$ will measurably reduce blood lead concentrations. It does not say that this reduction in blood lead would be permanent or cost-effective.

From another perspective, decisions about soil abatement are likely to be made on an individual home basis or on a neighborhood basis. For an individual home, the owner or renter may require only peace of mind in knowing that the property is safe for children if the soil lead concentrations are below an acceptable level, or, if not, that soil abatement would be a cost effective way to reduce or eliminate the problem.

This project shows that, on an individual house basis, soil abatement reduces the flow of lead into the home and its incorporation into house dust. The magnitude of this reduction depends on the concentration of lead in the soil, the amount of soil-derived dust that moves into the home, and the frequency of cleaning in the home. The number and ages of children and the presence of indoor/outdoor pets are factors known to increase this rate, whereas the frequency of cleaning with an effective vacuum cleaner and removing shoes at the door serve to reduce the impact of soil lead on house dust.

On a neighborhood basis, the focus of concern is usually directed at the local public health officer, who faces a risk management decision for which an exposure assessment based on the results of this project is only one element. Guidance in this case should provide general exposure scenario information that would assist the officer in predicting blood lead concentrations should soil be abated. There are many alternatives to soil abatement, such as control of pets, frequency of cleaning, or preservation of ground cover, available to the individual home owner or renter, that may not be practical for the public health officer making a decision on the neighborhood level.

# 2. BACKGROUND AND OVERVIEW OF PROJECT

## 2.1 PURPOSE OF THIS DOCUMENT

The purpose of this document is to describe the results of the Urban Soil Lead Abatement Demonstration Project (USLADP) from the perspective of all three studies that participated in the project. Taken individually, the reports of the three studies could support three different conclusions concerning the feasibility of reducing lead exposure by abating soil. Collectively, a common picture emerges that places a significant role for soil abatement in the total scheme of lead exposure reduction.

The purpose of USLADP was to determine if intervention in the form of soil abatement would reduce childhood exposure to lead. The project has taken nearly eight years from conception to completion. Each of the three studies in the project is a longitudinal study of the impact of an altered environment on the lead exposure of children. There are few other longitudinal studies of this type, and none of this scope or duration. Furthermore, the three studies were conducted using common protocols where possible, so that integrated analyses can broaden the base of information beyond the limits of a single study or location.

The project provides information for policy makers who need to recommend acceptable cleanup levels of lead in soil and dust that apply broadly to entire cities, states or the entire country, for public health officials who must provide guidance on site specific abatement decisions for individuals and families about the potential hazard on a single property, and for the scientific community whose job it is to challenge and refute incorrect information and to correct that information by further research.

For the policy maker, the Executive Summary (Chapter 1), provides a brief overview of the project, a short synopsis of the results, and a discussion of the recommendations and conclusions of the authors. For the Public Health Official who needs more detail in order to relate site specific situations to the generalized findings of this report, Chapter 2 provides a detailed description of the project and Chapter 5 gives an extended discussion of the conclusions and recommendations. A graphical presentation of the results may be found in Chapter 3 for the reader who seeks to understand in full detail the movement of lead in the human environment. These graphs, which systematically demonstrate the nature of this process, are supported by an array of statistical tests that are described in Chapter 4.

This document will reach its final form only after an extensive review process. First, the reports of the individual studies were reviewed by a panel of experts, revised, and presented to the U.S. EPA in their final form, along with the data sets that were used as the basis for the individual reports. These data sets were reanalyzed by EPA using rigorous statistical techniques to extract information not easily accessible with a single data set, and the integrated report, this document, was written based on these analyses. Following internal review and revision, the integrated report will be released in draft form for public comment and external review. The report will become final after the comments from the external panel of experts have been addressed and its release has been approved by senior EPA officials. At that time, members of the scientific community who have a legitimate research interest in the analysis of the data can obtain a copy of the data set for continued review and analysis.

Although the three studies were conducted independently, an effort was made to coordinate the critical scientific aspects of each study in order to provide comparable data at their completion. This effort included several workshops where the study designs, sampling procedures, analytical protocols, and QA/QC requirements of each study were discussed with a goal toward reaching a common agreement. In most cases, a consensus was reached on the resolution of specific issues, but the individual studies were not bound to conform to that consensus or to adhere to it throughout the study. Therefore, some attention will be given in this section to the differences in study design and experimental procedures among the three individual studies.

The results of these projects were presented at a symposium in August 1992. These presentations included the data analysis and conclusions of the three individual studies. Following this open discussion with the scientific community, the three groups submitted their respective reports to the designated U.S. Environmental Protection Agency (EPA) Regional Offices (Boston, Region I; Baltimore, Region III; and Cincinnati, Region V). These reports and their associated data sets were passed on to EPA/Office of Research and Development and EPA/Office of Solid Waste and Emergency Response (OSWER) for the preparation of this integrated report. Although it is unlikely that major findings have been overlooked in these first two phases, it is not at all unreasonable that more detailed information will be retrieved and reported by the extended investigations made possible by this open policy for data release.

This report presents the results and conclusions of the three studies in a manner in which they can be compared, both through a broad overview of the general findings and through the benefit of a detailed statistical reanalysis of the data. Because of the common design of the three studies and their focus on key experimental parameters, it is possible to combine the data for certain key parameters into a single data set for meta-analysis, and the results of this meta-analysis are also presented in this report.

## 2.2 PROJECT BACKGROUND

The Urban Soil Lead Abatement Demonstration Project, known also as the Three City Study, was authorized in 1986 under Section 111(b)(6) of the Superfund Amendments and Reauthorization Act (SARA). The scientific evidence for a correlation between soil lead and blood lead was sufficient to warrant concern for the health of children, but not strong enough to support a large scale program for soil lead abatement. Consequently, SARA (1986) called for EPA to conduct a "pilot program for the removal, decontamination, or other actions with respect to lead-contaminated soil in one to three different metropolitan areas."

To fulfill this mandate, it was necessary to design a project that would measure the change in exposure from a single source (soil) amid continual changes in exposure to other sources (air and food) and demographically irregular exposure from still more sources (drinking water and paint). Furthermore, the range of exposure for a 6-year-old child is much more diverse than for a 1-year-old child. Consequently, it was necessary to monitor all sources of lead and to include the abatement of entire neighborhoods as well as single residences.

### 2.2.1 Historical Perspective

In the past 25 years, concern for children with lead poisoning has steadily increased with mounting evidence for the subtle but serious metabolic and developmental effects of lead exposure levels previously thought to be safe. Childhood lead poisoning was formerly considered a severe medical problem usually traced to swallowed chips of peeling lead-based paint. Scientific evidence has systematically revealed deleterious effects of lead at lower levels of exposure, and regulatory agencies such as EPA and the Centers for Disease Control (CDC), have repeatedly lowered the level of concern for children's lead burden that requires environmental or clinical intervention. Whereas this concern was initially directed toward symptomatic children with blood lead levels of 60 $\mu$g/dL and above, since November 1991, lead poisoning has been defined by CDC as a blood lead level of 10 $\mu$g/dL or greater.

Children are exposed to lead through complex pathways from multiple sources. In the mid 1980s, attention to sources of childhood lead exposure turned to urban environments with high concentrations of lead in soil. Evidence for this concern came from the apparent correlation between the incidence of high blood lead concentrations and high concentrations of lead in residential soils. At that time, there were several other sources of exposure that could potentially account for unusually high blood lead in a population of urban children. Among these were lead in the air (primarily from automobile emissions), lead in food (primarily from canned foods with lead soldered side seams), lead in drinking water (primarily from lead pipes or newly soldered copper pipes), and lead in paint. The lead in the soil was believed to be a mixture of lead from the atmosphere and lead from exterior paint. Regulations were in place that would remove lead from gasoline by the end of 1986, and there was a voluntary program among food processors to phase out cans with lead soldered side seams and use only cans without lead solder.

The concept of soil abatement was not new in 1986. Many residential (usually nonurban) areas were located near Superfund sites where lead was a major contaminant, and the decision to abate soil was usually based in part on the distribution of blood lead within the population of children. There was, however, limited experience on the effects of this abatement and little or no opportunity to followup with studies of the results.

These complexities, and the overwhelming magnitude of the implications of this project, made it necessary to plan a project that was as broad in scope as possible within the resources available. One important concern was that there is a difference between a population of children that is suddenly placed into a "clean" environment and one that has lived continuously in a clean environment. Because of the physiology of lead mobilization in body tissues, there is a difference between the rate of change in a population with increasing lead exposure and in one with decreasing exposure. In other words, the decrease in blood lead anticipated in this project was not expected to be at the same rate as the increase observed in other studies.

### 2.2.2 Site Selection

The site selection process began in December 1986 with the appointment of a steering committee to develop recommendations for implementing the SARA lead-in-soil demonstration project. A panel of experts was formed in March 1987 to set the criteria for selection of sites and the minimum requirements for a study at each site. It would be necessary to design three concurrent studies that were identical except for key parameters such as the scope of the abatement (neighborhood versus single residence) and amount of soil contamination. An early decision was that the options for soil abatement methods were limited because only excavation and removal had been used in similar programs. Therefore, there would be no attempt in the project to evaluate alternative methods of abatement because of limited time and resources. The panel met again in April 1987 to discuss technical issues and study designs and evaluate technical criteria for selection of urban areas as potential soil-lead abatement demonstration project sites. They established the following site selection criteria.

A. To be considered for selection, a metropolitan area *must* have:

1. Agreement by the appropriate EPA regional office to provide general project oversight, and to disburse the funds.

2. An established entity, preferably the state, documented as willing to be responsible for removing and disposing of lead contaminated soil. This includes identification of an appropriate facility within the state for disposal of the soil, facilitation of permits, community relations and education, and any other activities necessary to expeditiously provide for disposal.

3. The administrative infrastructure to carry out a large scale project. This includes a key government department with appropriate authority to coordinate the project, and generally includes active participation by the state, by community groups, and by all the different metropolitan departments with some responsibility for the project.

4. Access to scientific and medical expertise is needed. Scientific advice will be needed to ensure that sampling and analysis are properly conducted; medical care will be needed for children found to have lead toxicity.

5. Evidence that there are both children with elevated blood lead levels as defined by the CDC in its childhood lead screening guidelines, and soil in residential areas to be abated with lead levels of 1,500 μg/g or greater. It would be desirable for leaded paint to be established as a major contributor to the soil lead levels.

B. To be considered for selection, a metropolitan area *should* have:

6. A documented high incidence of children with elevated blood lead levels in the proposed study areas. This means that the municipality supports an active childhood lead screening program.

7. A pattern of high density population in study areas. The number of children available for evaluation as part of the project is important to the statistical validity of the study.

8. Availability of other sources of funding for portions of the project not funded by SARA (1986). Such items might include de-leading the outside of houses, or intensive interior vacuuming to remove residual leaded dust.

The Steering Committee approved Boston as the initial study site and issued a Request for Proposal (RFP) in the summer of 1987 for two additional sites. Additional proposals were received from five other metropolitan areas: Baltimore, Cincinnati, Minneapolis, Detroit, and East St. Louis. These were reviewed on December 3 and 4, 1987, by the Steering Committee with additional experts. Baltimore and Cincinnati were selected to participate with Boston in the project. The following points were the basis for this decision.

1. Cincinnati proposed a neighborhood level abatement study where housing units had been previously gutted and rehabilitated approximately 20 years ago, and were considered free of lead-based paint.

2. The Cincinnati sites contained soil lead from 220 to 900 μg/g, exterior surface dust (primarily from paved areas) from 2,000 to 5,000 μg/g, and a number of children with blood lead concentrations above 25 μg/dL.

3. The Baltimore project proposed individual housing units with soil lead concentrations in excess of 10,000 μg/g. Lead-based paint had been abated in some, but not all houses.

4. There were few children in Minneapolis with blood-lead concentrations above 25 μg/dL, and most of the soil lead concentrations were below 1,000 μg/g.

5. Cincinnati demonstrated a high degree of organizational infrastructure, with commitments from the City of Cincinnati and the University of Cincinnati. There was an established structure for neighborhood associations that was perceived to be a plus for the project.

6. The Baltimore proposal was prepared by the State of Maryland and showed a satisfactory level of organizational infrastructure and local scientific expertise. There were problems noted with the statistical approach.

7. Although Detroit and East St. Louis both demonstrated significant lead problems through the numbers of children with elevated blood lead and the incidence of high soil lead, the organizational infrastructure and local scientific expertise in Detroit and East St. Louis were not perceived to be strong enough to support a project of this magnitude.

The selection panel was unanimous in their opinion that Baltimore and Cincinnati were the best choices to complement the Boston study, although they clearly recognized that Detroit and East St. Louis had a significant problem with urban soil lead. The panel believed that, if the study were conducted in Minneapolis where the soil lead concentrations were much lower than the other cities, the results would be too subtle to provide the level of statistical significance required for the study.

With the selection of Boston, Cincinnati, and Baltimore, a Scientific Coordinating Committee, with representatives from the three studies, the three EPA Regional Offices, OSWER, the Environment Criteria and Assessment Office/Research Triangle Park, NC (ECAO/RTP), and the Centers for Disease Control, was established to provide scientific and technical support for the three studies and to coordinate the exchange of scientific information. At an early stage, it was decided that the individual reports of the three studies, although published independently, would be combined into a comprehensive report that would seek to extract significant information that might not be otherwise available from the individual studies. The task of organizing the Scientific Coordinating Committee and writing this integrated report was assigned to ECAO/RTP. Major policy decisions remained with the Steering Committee.

Following the selection of Boston, Baltimore, and Cincinnati as the three sites for the project, the funding mechanisms were set into place individually through the respective EPA Regional Offices (Regions I, III, and V). Each of these regional offices set up an independent funding mechanism and oversight plan. The regional project officer became the liaison to the Steering Committee and to the Scientific Coordinating Committee. Each city submitted a work plan, which included the project description, organization, operation plan, and reporting mechanisms, and the Q/A plan. These work plans required more than one year to complete and pass Regional approval. In the meantime, the projects were staffed and became operational. Community relations programs were initiated that began the process of recruiting the study participants. Coordination between the three cities was accomplished through a series of workshops, approximately three per year, that continued through the preparation of the final reports.

## 2.3 PROJECT OVERVIEW

To place this project in perspective, it is helpful to look at the similarities and differences among the three studies. They are similar in that their hypotheses and study designs were drawn from the same general hypothesis, that removing lead from soil will reduce lead exposure. In a general sense, each study was designed around the concept of participating families within a definable neighborhood. There were one to three neighborhoods in each study group, and two or three study groups in each study. For each study group, there was a preabatement, abatement, and postabatement phase. This means that the environment of the child was extensively evaluated prior to and after abatement, through measurements of lead in soil, dust, drinking water, and paint, and through questionnaires about activity patterns, eating habits, family activities, and socioeconomic status. The objective of the preabatement phase was to achieve a clear understanding of the exposure history and status (stability of the blood lead and environmental measures) prior to abatement. During the abatement phase, attention was given to preventing any possible exposure that might result from the abatement activities. During the postabatement phase, the project was designed to determine the duration of the effect of soil abatement and to detect possible recontamination.

The project objective was to measure the relationship between soil lead and blood lead. This is an indirect relationship in the sense that children most commonly do not eat soil directly but usually ingest small amounts of dust derived, in part, from this soil. Likewise, the lead in blood reflects not only exposure from all sources, but a host of physiological processes that include distribution and redistribution of lead to other body tissues, especially bone tissue.

Throughout all phases, the timing of the blood lead measurements was in the context of a seasonal cycle of blood lead levels that peaks in the late summer and the age-related pattern that peaks at 18 to 24 mo. Because of the complex nature of this exposure assessment, it was deemed advisable to measure intermediate exposure indices, such as street dust, house dust, and hand dust, wherever possible. This required the development of new sampling and analysis protocols that were not generally available in the scientific literature, and thus became a major topic during the early coordinating workshops.

The studies differ in several respects. The pathways of soil to exterior dust and paint to house dust differ slightly among the studies, as do the intervention strategies to interrupt the flow of lead along these pathways. Collectively, these differences in study design broaden the scope of the project to cover aspects of lead exposure intervention not possible through the study of a single neighborhood or even a single city.

### 2.3.1 Project Terminology

The reader will more easily understand the discussions in this report if some consistency is given to certain descriptive terms that are found in the reports of the individual studies. These terms are described in the glossary of this document. An obvious example is the use of the terms "study" and "project". In order to avoid confusion, the term "study" refers to one of the three individual studies, and the term "project" is used in reference to the three studies collectively.

The names that were used by each study to identify the treatment groups have been modified in this report to assist the reader in remembering the type of intervention performed on each group. Table 2-1 lists these names, with a brief description and the corresponding term in the report of the individual study. The revised group names are linked to the location of the study and the nature of the intervention. For example, BOS SPI refers to the Boston group that received Soil, Paint, and Interior dust intervention. A hyphen is used to show that the intervention was separated by a period of time, as in CIN I-SE, where interior dust abatement took place about 1 year before soil and exterior dust abatement. The reader may want to become familiar with this nomenclature for the eight groups of participants in

TABLE 2-1. TREATMENT GROUP NOMENCLATURE WITH CROSS-REFERENCE TO INDIVIDUAL REPORTS

| Treatment Group Name[a] | Cross-Reference to Individual Study Report | Description of Treatment |
|---|---|---|
| | | BOSTON |
| BOS SPI | Study Group | Soil and interior dust abatement, and exterior and interior paint stabilization at beginning of first year, no further treatment |
| BOS PI | Control Group A | Interior dust abatement and exterior and interior paint stabilization at beginning of first year |
| BOS P | Control Group B | Exterior and interior paint stabilization at beginning of first year |
| | | BALTIMORE |
| BAL SP | Study Area | Soil abatement and exterior paint stabilization at beginning of first year, no further treatment |
| BAL P-1[b] | Control Area | Exterior paint stabilization at beginning of first year, no further treatment |
| BAL P-2[b] | Study Area | Exterior paint stabilization at beginning of first year, soil not above cutoff lead, no further treatment |
| | | CINCINNATI |
| CIN SEI | Area A | Soil, exterior dust, and interior dust abatement at beginning of first year, no further treatment |
| CIN I-SE-1[c] | Area B, Back Street and Findlay neighborhoods | Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment |
| CIN I-SE-2[c] | Area B, Dandridge neighborhoods | Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment |
| CIN NT | Area C | No treatment, soil and interior dust abatement at end of study |

[a]The treatment group designation indicates the location of the study (BOS = Boston, BAL = Baltimore, CIN = Cincinnati), the type of treatment (S = soil abatement, E = exterior dust abatement, I = interior dust abatement, P = loose paint stabilization, NT = no treatment).
[b]Treated as one group in the Baltimore report, analyzed separately in this report.
[c]Treated as one group in the Cincinnati report, analyzed separately in this report.

the project, as the data and results will be presented using these designations without further explanation. One further note: The BOS PI and BOS P groups each received soil abatement at the end of the study. Because no data were reported following this intervention, the designation "-S" was not used in order to avoid confusion.

Other departures from the terminology of the respective study reports are conversion to a common system of units (metric where possible) and standard terms for phases, stages, or rounds of the project. In the latter case, the term "Round" is used for the study phase, and there is no consistent pattern for when abatement occurs (i.e., after Round 1, Round 3, etc.).

### 2.3.2 Study Groups

Variations in the nature and form of intervention were included in the study designs, such that specific types of information could be retrieved from individual studies based on the unique characteristics of the cities and their neighborhoods. For example, neighborhoods in Cincinnati were selected because they were relatively free of lead-based paint, a known confounder in the relationship between soil lead and blood lead. As it happened, these neighborhoods were mostly multifamily housing with little or no soil on the residential parcel of land. The study design used intervention on the neighborhood scale, where the soil in parks, play areas, and other common grounds could be abated, and paved surfaces in the neighborhood could be abated of exterior dust lead. Table 2-2 describes the study design characteristic for each of the three studies and their respective neighborhood groups. The general characteristics are that soil lead concentrations are typically high in Boston, where it is also common to find lead in drinking water and in both exterior and interior paint. In the areas studied, housing is typically single family with relatively large soil cover. In the Baltimore neighborhoods, nearly every house had lead-based paint, the houses were mixed single and multifamily, and the soil areas were smaller, typical less than one hundred square meters.

### 2.3.3 Intervention Strategies

Intervention is defined here as the interruption of the flow of lead along an exposure pathway. Soil abatement is one form of intervention. If done correctly, this abatement should establish an effective and persistent barrier to the dust movement. Other forms of

## TABLE 2-2. NUMBER OF PROJECT PARTICIPANTS BY STUDY GROUP AND ROUND[a]

| | Study Group | | | | | | |
|---|---|---|---|---|---|---|---|
| BOSTON | | R1 | R3 | R4 | | | |
| Middate | | 10/17/89 | 4/9/90 | 9/12/90 | | | |
| Children[b] | BOS SPI | 52 | 52 | 52 | | | |
| | BOS PI | 51 | 48 | 49 | | | |
| | BOS P | 47 | 46 | 46 | | | |
| Famlies[c] | BOS SPI | 43 | 43 | 43 | | | |
| | BOS PI | 43 | 40 | 41 | | | |
| | BOS P | 39 | 38 | 38 | | | |
| Properties[d] | BOS SPI | 34 | 34 | 34 | | | |
| | BOS PI | 36 | 33 | 34 | | | |
| | BOS P | 30 | 29 | 29 | | | |
| BALTIMORE | | R1 | R2 | R3 | R4 | R5 | R6 |
| Middate | | 10/25/88 | 4/1/89 | 2/17/90 | 1/27/91 | 6/7/91 | 9/3/91 |
| Children[b] | BAL SP | 75 | 70 | 95 | 89 | 85 | 81 |
| | BAL P | 333 | 252 | 174 | 111 | 111 | 106 |
| Families[b] | BAL SP | 53 | 43 | 32 | 23 | 22 | 21 |
| | BAL P | 210 | 160 | 125 | 93 | 88 | 88 |
| Properties[b] | BAL SP | 48 | 44 | 51 | 49 | 45 | 45 |
| | BAL P | 213 | 157 | 105 | 65 | 63 | 63 |
| CINCINNATI | | R1 | R3 | R5 | R7 | R9 | |
| Middate | | 7/6/89 | 11/14/89 | 7/1/90 | 11/17/90 | 6/16/91 | |
| Children[b] | CIN SEI | 54 | 52 | 46 | 37 | 31 | |
| | CIN I-SE | 86 | 81 | 92 | 87 | 77 | |
| | CIN NT | 61 | 52 | 81 | 74 | 61 | |
| Families[c] | CIN SEI | 31 | 30 | 31 | 31 | 30 | |
| | CIN I-SE | 58 | 56 | 56 | 74 | 60 | |
| | CIN NT | 40 | 37 | 35 | 63 | 52 | |
| Properties[d] | CIN SEI | 55 | 39 | 39 | 40 | 40 | |
| | CIN I-SE | 74 | 121 | 121 | 119 | 121 | |
| | CIN NT | 86 | 85 | 85 | 84 | 84 | |

[a]Round designations (R1, R2, etc) are the same as used in the individual study reports. Some rounds are omitted from this table because no participant data were collected. Intervention occurred between R1 and R3 in Boston, R3 and R4 in Baltimore, R1 and R3 in the first year of the Cincinnati study, and R5 and R7 in the second year.
[b]Based on number of children sampled for blood.
[c]Based on number of households sampled for dust.
[d]Based on number of soil areas sampled.

intervention used in this project were exterior dust abatement, interior dust abatement, and paint stabilization. Because dust is a very mobile constituent of the human environment, exterior and interior dust abatement would not be expected to form a permanent barrier to lead unless other sources of lead, such as soil, were also abated.

Figure 2-1 illustrates the generalized concept of the pathway and sources of human exposure to lead, showing the routes of lead from the several sources in the human environment to four compartments (inhaled air, dusts, food, drinking water) immediately proximal to the individual. One of these proximal sources, dust, is the primary route of concern in this project. Figure 2-2 expands this dust route to show the complexity of the many routes of dust exposure for the typical child.

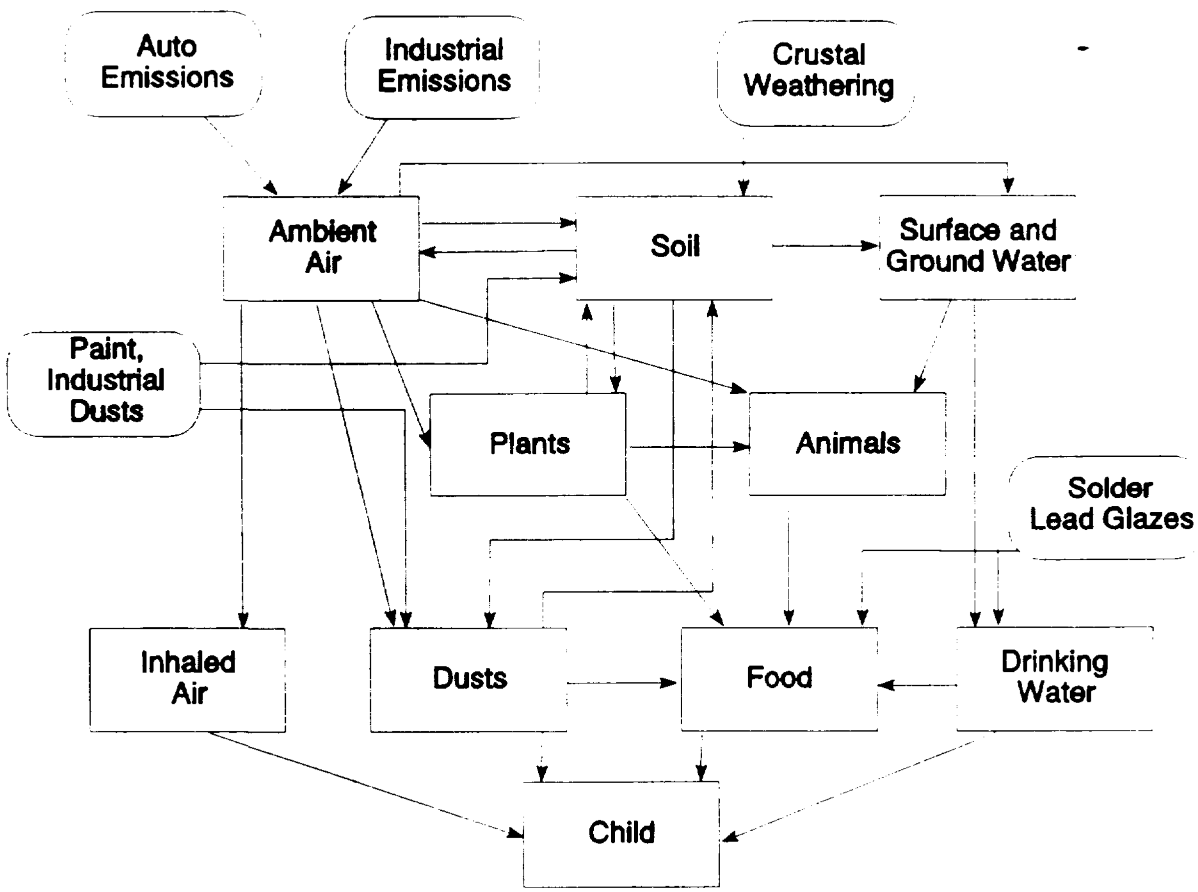


**Figure 2-1. Generalized concept of the sources and pathways of lead exposure in humans.**

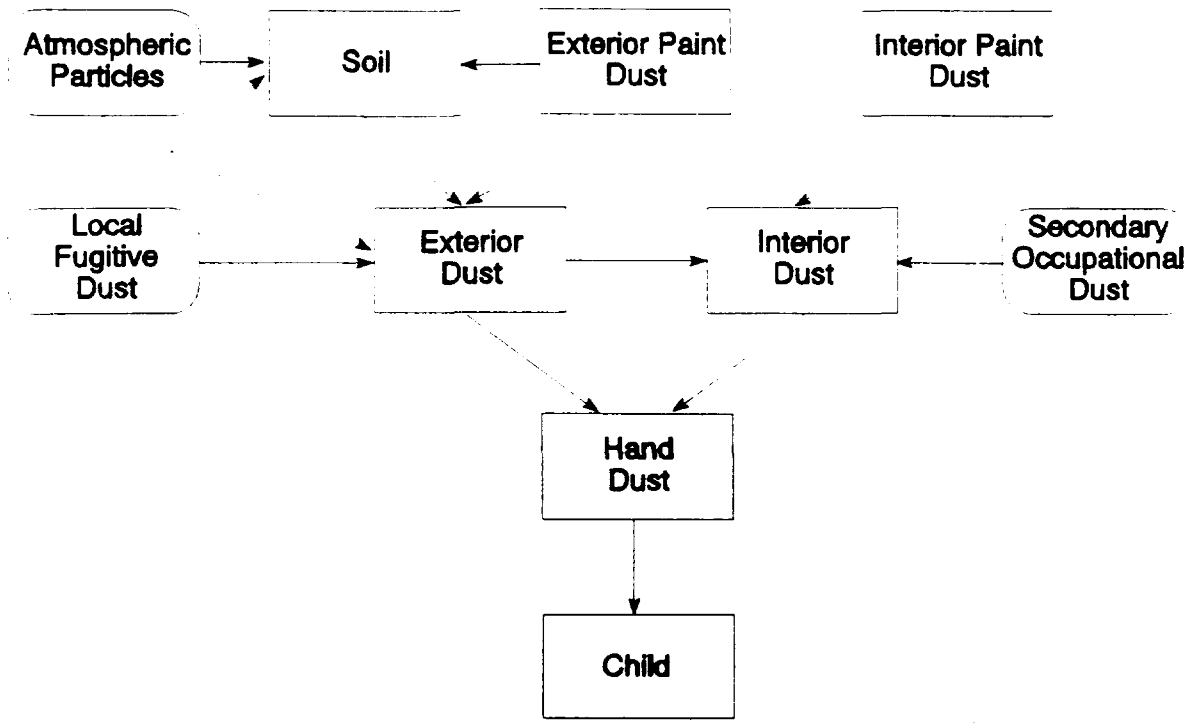


**Figure 2-2. Typical pathways of childhood exposure to lead in dust.**

The strategies for intervention used in this project were designed to interrupt the movement of lead along one or more of these pathways. The pattern for these interventions for each study appears on Figures 2-3, 2-4, and 2-5.

The strategy for soil abatement was to remove all soil with concentrations above a specific level (determined for each study), and replace this soil with clean soil below a specified lead concentration. This method, called excavation and removal, was used in all three studies. In some cases, repair and maintenance of ground cover was used where the soil concentrations did not warrant excavation and removal.

To further interrupt the flow of lead along the exposure pathways, entire neighborhoods were cleaned of exterior dust using vacuum equipment and hand tools. This intervention method was used only in Cincinnati.

Interior dust abatement was expected to have an immediate effect on the blood lead of children because household dust is believed to be their primary source of lead. Because household dust is a mixture of several sources of lead, abating house dust temporarily removes these sources, and their return and consequent impact on the child's environment

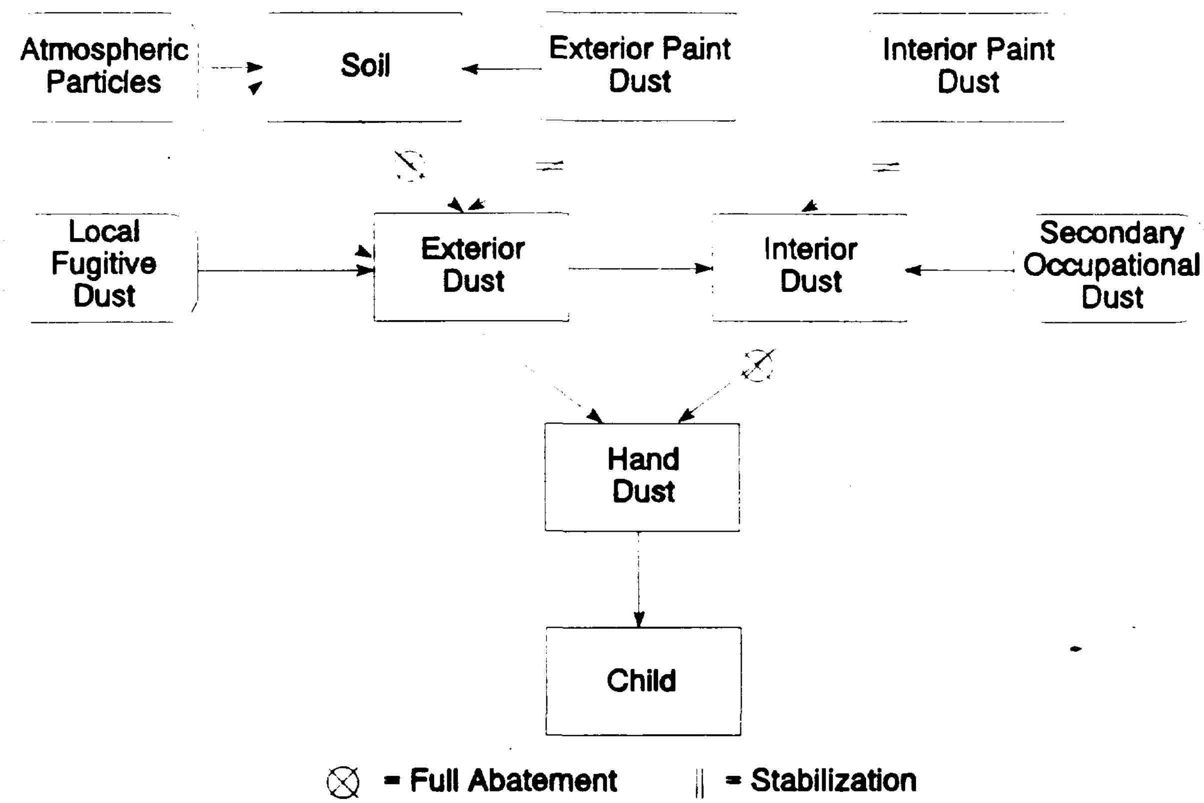


**Figure 2-3. Pathway intervention scheme for dust exposure (Boston Soil Abatement Study).**

can be evaluated by careful measurements of the household dust. It is essential that information about the change in lead concentration, lead loading, and dust loading be a part of these measurements. Following dust abatement, there should be an immediate decrease in the dust loading, with no change in the lead concentration for those groups that did not receive soil, exterior dust, or paint intervention. The rate at which this dust loading returns to preabatement levels reflects the rate of movement of dust from other sources into the home and the general housekeeping effectiveness of the home. Both of these factors are believed to have some influence on the amount of dust that a child ingests.

The effectiveness of both paint stabilization and soil and dust abatement can be observed by changes in the lead concentrations of house dust. In the presence of lead-based paint, the concentration of lead in house dust is expected to be greater than 1,500 to 2,000 $\mu$g/g, whereas without the influence of lead-based paint, the house dust is expected to be comparable to external dust and soil (U.S. Environmental Protection Agency, 1986).

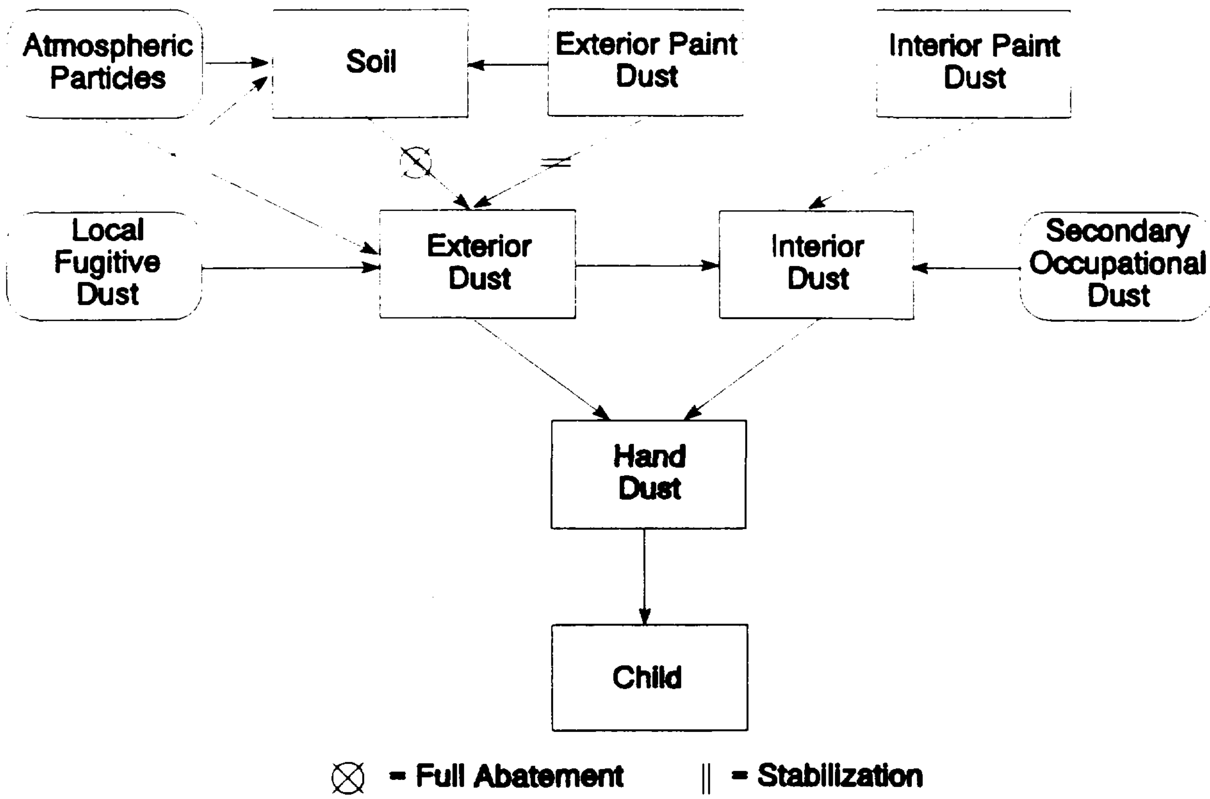


**Figure 2-4. Pathway intervention scheme for dust exposure (Baltimore Soil Abatement Study).**

House dust is a mixture of dusts from many sources within and outside the home. In the absence of lead-based paint inside the home, it would seem reasonable to assume that most of the lead in household dust comes from soil and other sources immediately external to the home. Therefore, to enhance the impact of soil abatement, interior dust abatement was carried out for some study groups in Boston and Cincinnati, but not in Baltimore.

Many of the Boston and Baltimore households selected for the project had chipping and peeling paint, both interior and exterior. In order to reduce the impact of this paint, much of which was lead-based paint, the walls and other surfaces were scraped and smoothed and repainted to reduce the impact of lead-based paint on the pathways of lead exposure. It is important to note that this approach in not a full scale paint abatement and was not designed to place a permanent barrier between the paint and the child. Paint stabilization was used on exterior and interior surfaces in Boston and on exterior surfaces in Baltimore. Paint

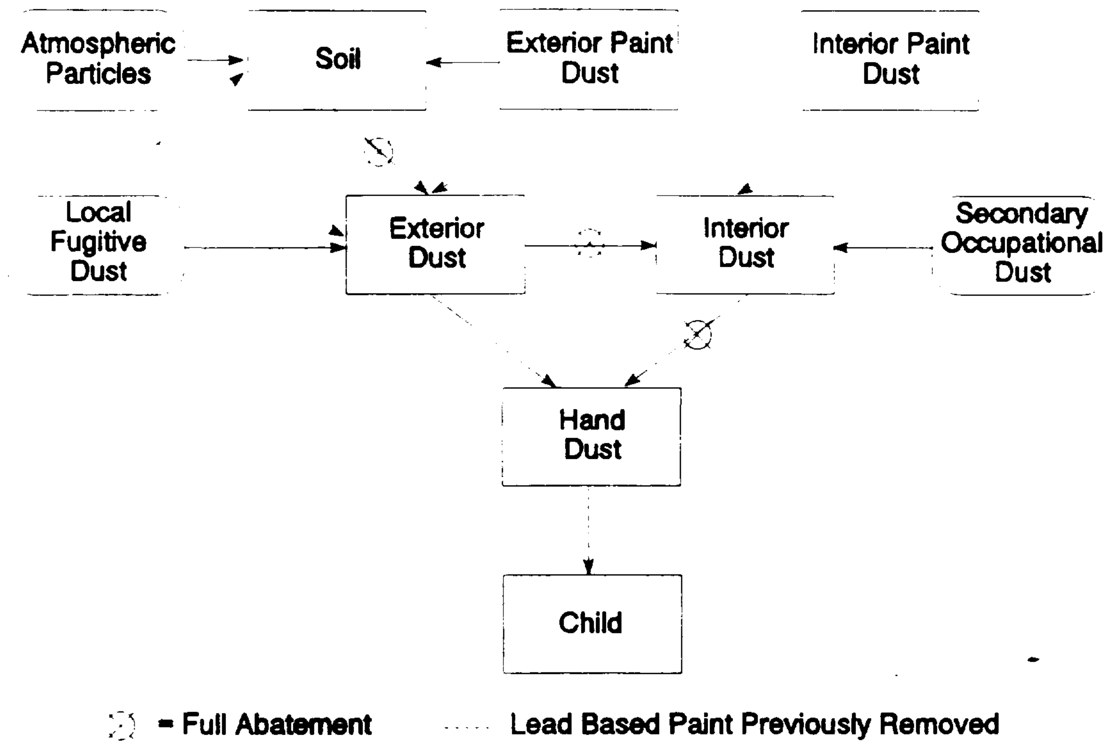


**Figure 2-5. Pathway intervention scheme for dust exposure (Cincinnati Soil Abatement Study).**

stabilization was not used in Cincinnati because the lead-based paint had been removed from these homes in the early 1970s.

### 2.3.4 Measurements of Exposure

Exposure is the amount of a substance that comes into contact with an absorbing surface over a specific period of time. In the case of lead, the absorbing surface can be the gastrointestinal tract or the lungs. Exposure is measured in $\mu$g/day. Thus, an exposure of 10 $\mu$g/day represents a total ingestion and inhalation of 10 $\mu$g lead from all sources; a fraction of this 10 $\mu$g would be absorbed into the body. In this project, blood lead was used as an indicator of exposure, and reductions in blood lead were expected as a result of any combination of the interventions described above. The units are $\mu$g Pb/dL blood, and they are not compatible with the normal units of exposure, $\mu$g Pb/day. This illustrates that lead in 1 dL of blood reflects the accumulation of an unknown number of days. Other measures of exposure are hand lead and house dust. The amount of lead on the child's hands is believed to be closely related to the blood lead. House dust can be an effective estimator of exposure when then child spends most of the day playing inside, or when the house dust is composed largely of exterior dust.

#### 2.3.4.1 Blood Lead

The amount of ingested lead that is actually absorbed depends on the bioavailability of the particular form of lead. The amount of absorbed lead that reaches specific body tissues depends on the biokinetics of lead in the human body. Because there is also a relationship between blood lead and the onset of health effects of lead, it is convenient for this measure of blood lead to be used both as an indicator of exposure and a measure of the potential health risk to the child. Blood tissue is in dynamic equilibrium with all other body tissues, including bone tissue, where the lead is stored for longer periods of time. This situation becomes important when measuring the rate at which blood lead concentrations might decline following abatement. For a child with lead stored in bone tissue following a long history of lead exposure, the decline in blood lead might be expected to be slower than a child without previous exposure.

#### 2.3.4.2 Hand Lead

Because blood lead reflects exposure to lead from all environmental sources, a second measure, hand lead, was used to focus directly on the immediate pathway of dust into the child. The units of measure are $\mu$g Pb/pair of hands, and like blood lead, this measure does not reflect the rate at which lead moves to the body in the form of $\mu$g Pb/day. Instead, this hand dust is a measure of lead loading. It is a measure of the "dirtiness" of the hand in the same sense that dust loading is a measure of the dirtiness of the floor, as discussed in the next section. Hand dust loading could be converted to $\mu$g/day if there were a measure of the number of "hands" (or hand area) mouthed by the child during one day.

#### 2.3.4.3 House Dust

House dust is a mixture of lead from many sources, including soil, street dust, interior paint, and several biological sources such as insects, pets, and humans. The units of measurement are $\mu$g Pb/g (lead concentration), $\mu$g Pb/m$^2$ (lead loading), and mg dust/m$^2$ (dust loading). When expressed as $\mu$g Pb/g, the measurement can be converted to an exposure measurement by assuming a specific amount of dust ingested per day, usually about 100 mg/day for preschool children. Exposure to household dust then becomes $\mu$g/day:

$$\text{Pb Concentration} \times \text{Ingestion} = \text{Exposure}$$

$$\frac{\mu g Pb}{g\,dust} \times \frac{g\,dust}{day} = \frac{\mu g Pb}{day}. \tag{2-1}$$

To understand the importance of this measurement, compare the exposure from household dust to other sources. Food, drinking water, and inhaled air normally account for about 5 to 15 $\mu$g Pb/day. If the lead concentration in household dust is 200 $\mu$g/g and dust ingestion is 0.1 g/day, the exposure is 20 $\mu$g/day or more than the other sources combined. In this project, the maximum lead concentration household dust was 107,000 $\mu$g/g.

House dust can be an important measure of potential lead poisoning, and abatement efforts, whether soil or paint abatement, will not be successful unless there is a reduction in exposure to lead in house dust.

## 2.3.5 Treatment Approaches

### 2.3.5.1 Soil Abatement Approaches

The approach to soil abatement used in Boston was to remove the top 15 cm of soil, apply a synthetic fabric, and cover with a layer of about 20 cm of clean topsoil. The new soil was covered with sod and watered through dry months. Areas not resodded were covered with a bark mulch. Some driveways and walkways were covered with 5 cm soil and 15 cm gravel or crushed bank (stone with dust). On four properties, the driveway was capped with 7.5 cm asphalt without soil removal, at the owner's request. A total of 93 Boston properties were abated in this manner. The information on area treated and volume of soil removed from these properties appears in Table 2-3. The method of excavation was by small mechanical loader (Bobcat) and hand labor, for the most part. Initially, six properties were abated with a large vacuum device mounted on a truck, but this proved unsatisfactory due to the size and lack of maneuverability. During one extreme cold

TABLE 2-3. SOIL ABATEMENT STATISTICS FOR THE THREE STUDIES

| | Boston | Baltimore | Cincinnati |
|---|---|---|---|
| Number of properties[a] | 36 | 63 | 171 |
| Surface area ($m^2$) | 7,198 | 4,100[b] | 12,089 |
| Volume soil removed ($m^3$) | 1,212 | 690 | 1,813 |
| Surface area/property ($m^2$) | 200 | 73 | 70.7 |
| Volume soil/property ($m^3$) | 33.7 | 10.9 | 11 |

[a]Includes only properties abated during study. Properties abated at the end of the study, where no further sampling was reported, are not included in this analysis, but are included in the individual study reports. In Cincinnati, a property is the location of the soil abatement, not the location of the child's residence.
[b]Surface area not provided by Baltimore report. Calculated using Boston vol/surf ratio, which is equivalent to an average removed depth of 17 cm.

spell, it was necessary to remove large blocks of frozen soil, often greater than 15 cm thick, by loosening with a jackhammer.

In Baltimore, 63 properties in BAL SP were abated between August and November 1990. An additional seven properties that did not meet the requirements for abatement were transferred to the control group (BAL P). Unpaved surfaces were divided into areas on each property, usually front, back, and one side; and any area with soil lead concentrations above 500 $\mu$g/g was abated entirely. Soil and ground cover were removed down to 15 cm and replaced to the original level with soil lead concentrations less than 50 $\mu$g/g. These areas were sodded or reseeded as appropriate. Bare areas were prepped and reseeded even if soil lead concentrations did not warrant excavation. Additional information appears in Table 2-3.

Within each neighborhood, the Cincinnati study identified all sites with soil cover as discrete study sites. The decision to abate was based on soil lead concentrations for each parcel of land, and for the depth to which the lead had penetrated. Lead was measured in the top 2 cm and at a depth of 13 to 15 cm. If the concentration in the top sample was greater than 500 $\mu$g/g, the soil was abated. For areas where the top concentration was less than 500 $\mu$g/g but the profile (mean of top and bottom) was between 300 and 500 $\mu$g/g, the soil was resodded if bare. Initially, there was an option to cultivate by rototilling, but this approach was abandoned as not feasible in this study. Excavation was by front end loader, backhoe and hand tools down to 15 cm, and the replacement soil lead concentration was less than 50 $\mu$g/g. Further information can be found in Table 2-3.

#### 2.3.5.2 Exterior Dust Abatement Approach

Exterior dust abatement was performed in the Cincinnati study only. The approach to this abatement was to identify all types of paved surfaces where dust might collect, obtain permission to sample and abate these areas and to clean them once with vacuum equipment, suitable for the area, that had previously been tested and shown to remove about 95% of the available dust on the area. The groups of surfaces selected were streets, alleys, sidewalks, parking lots, steps, and porches. For data analysis, these were grouped as (1) targeted (steps and porches); (2) streets, sidewalks, and alleys; and (3) parking lots and other locations.

If it were true that soil is the only source of lead in the urban neighborhood, then analysis of external dust would provide a measure of the movement of lead. In the case where the soil was abated, then external dust abatement would speed up the rate at which the effectiveness of this abatement could be seen on the interior dust of homes. Where the soil was not the only source of lead, the recontamination of exterior dust might shed some light on the movement of lead from other sources onto the hard surfaces that characterize child playtime activities.

The exterior dust measurements in the Cincinnati study (and the interior dust measurements of all three studies) were made in a manner that determined the lead concentration ($\mu$g Pb/g dust), the dust loading (mg dust/$m^2$), and the lead loading ($\mu$g Pb/$m^2$) for the surface measured. This required that a dry vacuum sample be taken over a prescribed area, usually 0.25 to 0.5 $m^2$. It is important to note that dust abatement is not expected to cause an immediate change in the lead concentration on dust surfaces, only the dust and lead loading.

#### 2.3.5.3 Interior Dust Abatement Approaches

Household dust was abated in the Boston and Cincinnati studies, but not in Baltimore. The BOS SPI and CIN SEI groups received interior dust abatement at the same time as soil abatement, the BOS PI received interior dust abatement without soil abatement, and the CIN

I-SE received interior dust abatement in the first year followed by soil abatement in the second year.

In Boston, interior dust abatement was performed after loose paint stabilization. Families were moved offsite during interior dust abatement. Hard surfaces (floors, woodwork, window wells, and some furniture) were vacuumed with a High Efficiency Particle Accumulator (HEPA) vacuum, as were soft surfaces such as rugs and upholstered furniture. Hard surfaces were also wiped with a wet cloth (an oil treated rag was used on furniture) following vacuuming. Common entries and stairways outside the apartment were not abated.

The Cincinnati group performed interior dust abatement after exterior dust abatement and also moved the families offsite during this activity. Vacuuming of non-carpeted areas, which was done two times, each at a prescribed rate of 1 $m^2$/min, was followed by wet wiping with a detergent. They replaced one to three carpets and two items of upholstered furniture per housing unit. Their previous studies had shown that these soft items could not be cleaned effectively with vacuuming alone. Where carpets were left in the home, they were vacuum cleaned three times, each at a rate of 1 $m^2$/min.

Although it is clear that interior dust abatement had a positive effect on reducing the lead in the child's environment in the Boston study, the influence of paint stabilization (discussed below) on household dust must also be considered in conjunction with the impact of soil abatement.

#### 2.3.5.4 Loose Paint Stabilization Approaches

It was the intent of the project to maximize the impact of soil abatement by minimizing the influence of lead-based paint in all three studies. Most homes in the Cincinnati group had received paint abatement 20 years prior to the project, but in Boston and Baltimore, lead-based paint occurred in nearly every home. Because full paint abatement was not within the scope of this project, the alternative was to retard the rate of movement of paint from the walls to household dust to the extent possible. The interior and exterior surfaces of all Boston homes and the exterior surfaces of all Baltimore homes received loose paint stabilization at the beginning of the project, prior to any other intervention.

In Boston, loose paint stabilization consisted of removing chipping and peeling paint with a HEPA vacuum and washing the surfaces with a trisodium phosphate and water solution. Window wells were painted with a fresh coat of primer. The exterior painted surfaces of Baltimore homes were wet scraped over the chipping and peeling surfaces, followed by HEPA vacuuming. The entire surface was primed and painted with two coats of latex paint.

Although there were subsequent measurements made of the presence of lead-based paint, there were no measurements made of the movement of lead from paint to house dust that would reflect the effectiveness or persistency of paint stabilization. It was believed that any contamination from lead-based paint would be readily apparent in the dust samples, and this appears to be the case (Chapter 3).

### 2.3.6 Project Activity Schedule

The project activity schedule, shown in Figure 2-6, illustrates the major intervention and measurement activities of the individual studies and the sequence and duration of these activities. The frequency and timing of sampling relative to abatement and seasonal cycles are important issues in the study design. These time lines are the actual occurrence of these events and they differ somewhat from the planned schedule. The original design focused on sampling blood lead during the late summer, as it was known that the seasonal cycle for blood lead reaches a peak during this period. Where this schedule could not be adhered to, an effort was made to schedule the followup sampling to characterize this cycle and possibly permit extrapolation to the summer peaks.

### 2.3.7 Quality Assurance/Quality Control Plan

Each study established a sampling and analysis plan that included rigorous quality control and quality assurance (QA/QC) objectives. To achieve these objectives, protocols were developed to: (1) define sampling schemes that characterize the expected exposure to soil for children; (2) collect, transfer, and store samples without contamination; and (3) analyze samples with the maximum degree of accuracy and precision. Several intercalibration exercises were performed to guarantee that the analytical results for

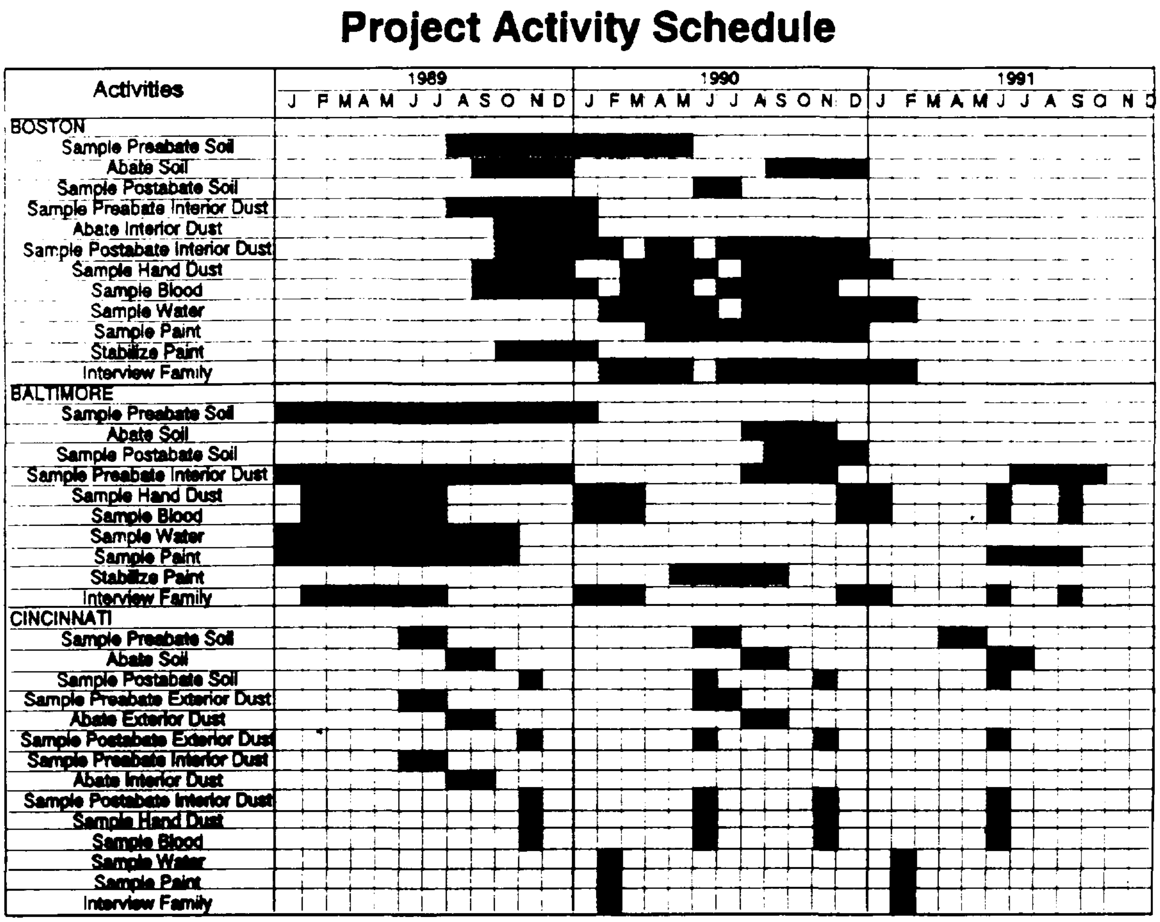


**Figure 2-6. Project activity schedule showing the times of sampling and interviewing (shaded bars) and soil abatement (solid bars).**

measurements of soil, dust, handwipes, and blood were accurate and that the data would be intercomparable.

### 2.3.7.1 Quality Assurance/Quality Control for Soils and Dusts

A major objective of the QA/QC program was to ensure that the three studies could achieve a comparable level of expertise in the analysis of soil samples. One measure of this expertise is whether the laboratories of the three studies would each get the same results when analyzing the same soil sample. Two round robin calibration exercises were conducted, one at the beginning and one near the end of the project. In each exercise, two additional laboratories were included in order to determine some measure of comparability with other studies reported in the scientific literature. All laboratories reported their results independently. In the interval between these two calibration exercises, double blind audit samples were inserted into the sample stream of each study to measure the persistency of analytical precision throughout the study.

*Round Robin Calibration Exercise I*

In May 1988, prior to the beginning of each study, each of the three laboratories collected ten soil samples from areas similar to those that would be included in their study. One of the samples from Cincinnati was a street dust sample of very high lead concentration. The other 29 samples were selected from soils with lead concentrations expected to range from 250 to 8,000 $\mu g/g$. The samples were dried and sieved according to the study protocols. Approximately 200 g of each sample were sent to the other two laboratories and to an outside lab at Georgia Tech Research Institute. Table 2-4 shows the instrumentation and method of analysis used by each laboratory. The results are presented in Chapter 3. In making these analyses, each laboratory used its own internal standards for instrumental calibration and shared a common set of five standards provided by Dr. Rufus Chaney at the U.S. Department of Agriculture. The intercalibration exercise successfully established a baseline for cross study comparison of soil and dust results.

**TABLE 2-4. WET CHEMISTRY AND INSTRUMENTAL METHODS USED FOR THE FIRST INTERCALIBRATION STUDY**

| | Participating Laboratories | | | | |
|---|---|---|---|---|---|
| Method[a] | Boston | Baltimore | Cincinnati | GTRI[b] | USDA[c] |
| Hot $HNO_3$/AAS | | X | X | | |
| Cold $HNO_3$/AAS | | | X | | X |
| Hot $HNO_3$/ICP | | X | | | |
| XRF | X | | | X | |

[a]$HNO_3$ = Nitric acid; AAS = Atomic absorption spectroscopy; ICP = Inductively coupled plasma emission spectroscopy; XRF = x-ray fluorescence.
[b]GTRI = Georgia Tech Research Institute.
[c]USDA = U.S. Department of Agriculture.

*Intercalibration Standards and Audits*

The first intercalibration exercise also revealed a need for a set of common standards that could be used to intercalibrate the laboratories and to monitor the performance of each laboratory throughout the project. The three studies each collected three soil samples in bulk (about 30 kg) in a range thought to be high, medium, and low for their area. The samples were dried, sieved, and analyzed at the EPA Environmental Monitoring Systems Laboratory in Las Vegas, NV (EMSL/LV). Following homogenization, approximately fifty samples of each of the soils were analyzed by laboratory scale X-ray fluorescence (XRF) at the EMSL/LV laboratory. Three of the nine soils were distributed to the participating cities for use as common external reference standards. The remaining six were used as double blind external audits. These were aliquoted into approximately 20-g samples, distributed to the QA/QC officer of each study, and inserted into the soil sample stream fully disguised as field soil samples.

Each city appointed a QA/QC officer who was not directly involved with the analysis of the soil samples, but who had access to the soil sample preparation stream on a daily basis. This person mailed prelabeled soil sample containers with typical sample numbers to the EMSL/LV laboratory. Soil aliquots typical for each city were placed in the sample containers and returned to the QA/QC officer in lots of 20 to 30. The identification numbers and soil concentration values were sent to the project QA/QC officer at ECAO/RTP. Each city's QA/QC officer inserted the double blind samples into the sample stream on a random basis at a frequency that would ensure about four QA/QC samples per analytical day. These were occasionally placed as duplicates in the same batch to provide information about replication within the batch.

The preliminary acceptance range for the double blind audit samples was established using only the 50 XRF analyses by the Las Vegas laboratory. As the analytical results were reviewed by the study QA/QC officer, the audit sample results were sent to the project QA/QC officer. If the audit samples were outside the acceptable range, the study QA/QC officer was informed and could recommend either reanalysis or flagging the data for that entire batch. In most cases the data were flagged, because the range for the six audit samples was very narrow, having been established based on analyses by a single laboratory (EMSL/LV). There was no allowance for interlaboratory variation. Final decisions on the disposition of the audit sample anomalies were deferred until the completion of the second intercalibration exercise near the end of the study, which provided a basis for determining the means and ranges for these audit samples.

*Round Robin Intercalibration Exercise II*

Near the end of the project, aliquots of the nine soil and six dust audit samples used during the project were redistributed to the three study laboratories for single blind analysis. The analyst was aware that the samples were audit samples, but did not know their concentrations. These measurements were the basis for establishing the final range of acceptability for the audit samples, and for correcting the soil and dust measurements to values common to the project.

### 2.3.7.2 Quality Control and Quality Assurance for Hand Dust

The collection and analysis of hand wipes is a new procedure developed just prior to the beginning of the project. There were few published reports of the measurement techniques, no certified standards, no internal standards, and little to base decisions on acceptable analytical precision. Double blind audit samples were provided the study QA/QC officer as an external control for hand wipe analysis. These were prepared as simulated samples by placing a known amount of an appropriate solution of lead nitrate solution onto the blank hand wipe at the EMSL/LV laboratory, wrapping and labeling according to the field protocol and returning to the participating laboratory for insertion into the sample scheme. There was no attempt to determine interlaboratory variance or to calculate correction factors. The study QA/QC officer was responsible for reporting problems to the laboratory director.

### 2.3.7.3 Quality Control and Quality Assurance for Blood Lead

The QA/QC program for blood analysis was directed by Dr. Dan Pascal of the Centers for Disease Control using the protocols developed for the CDC blood lead certification program. Each laboratory received double blind bovine blood samples with one of four lead concentrations. The range of acceptable measured concentrations was the same as for the certification program, and all three participating laboratories reported satisfactorily in this program.

## 2.3.8 External Factors That Could Influence the Outcome of the Project

The Scientific Coordinating Panel recognized several factors that might influence the outcome of the project and that were generally beyond the control of the investigators. Among these are seasonal cycles and time trends of childhood blood lead concentrations, unexplained or unexpected sources of lead in the homes or neighborhoods, changes in the public perception of the hazards of lead exposure and awareness of exposure reduction efforts, and movement of lead in soil either down the soil column or laterally as drainage or fugitive dust.

### 2.3.8.1 Cycles and Trends in Environmental Lead Concentrations

Investigators have known for some time that there is a seasonal pattern to the blood lead measurements taken for a population of children. Most epidemiological studies are planned so that measurements can be taken at the peak of this cycle, generally during the late summer. Prior studies of large numbers of children show a clear sinusoidal pattern, even when the measurements do not include sequential measurements for the same child. During the development of the study designs, it was apparent that an understanding of the seasonal cycles and temporal trends in blood lead would play an important part in the interpretation of data collected over several years. Many earlier studies had shown a clear seasonal cycle, with a peak in late summer, for the blood lead concentrations of urban children. Figure 2-7 illustrates this pattern for Chicago during the 1970s, at the same time showing a downward trend throughout the decade. The National Health Assessment and Nutrition Examination Survey II (NHANES II) data for the entire country and all age groups (Figure 2-8) show a similar seasonal cycle and downward trend during the last half of that decade. Although this project was designed to maximize the measurements of blood lead during the late summer for each of the three studies, many measurements were made during other times of the year in order to observe changes immediately after abatement. Consequently, there are sequential measurements for a large number of children that should be adjusted for seasonal effects in order to interpret the response to intervention.

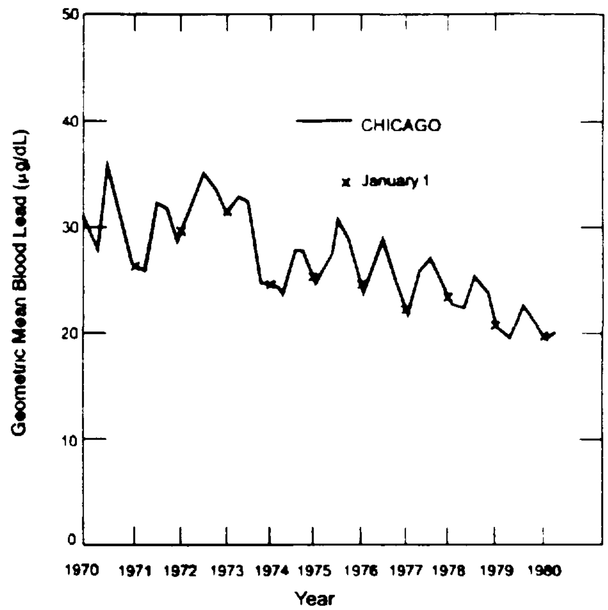


**Figure 2-7. Literature values for seasonal patterns for childhood blood lead (age 25 to 36 mo).**

Source: U.S. Environmental Protection Agency (1986).

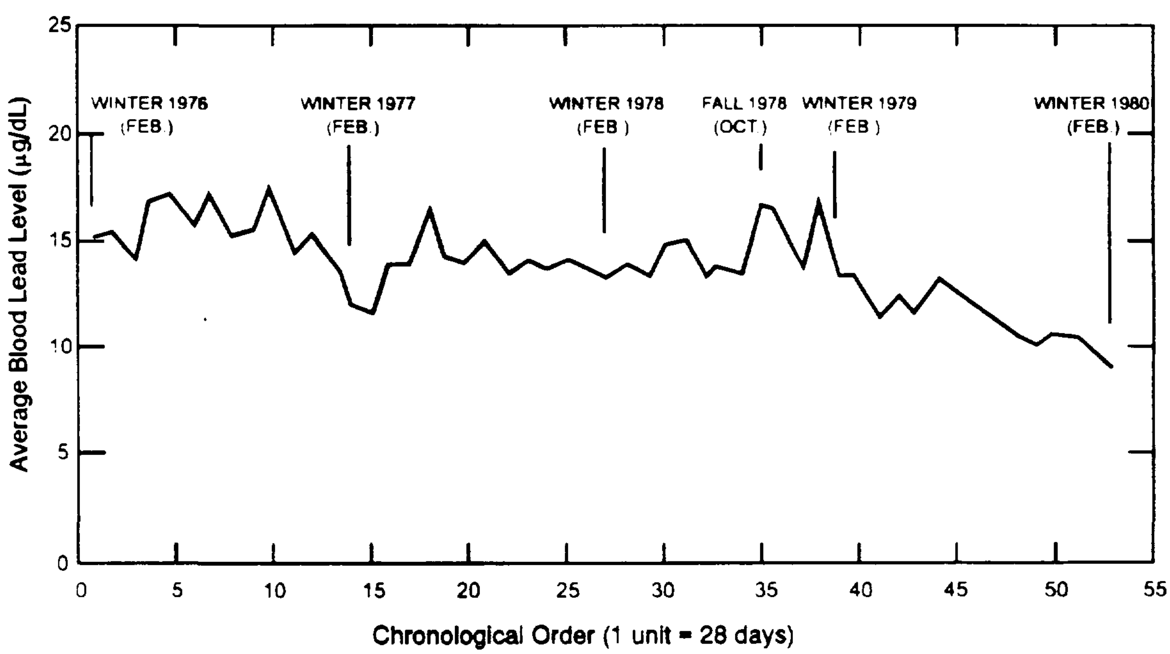


**Figure 2-8. Literature values for seasonal patterns for blood lead in children and adults (NHANES II, age 6 mo to 74 years).**

Source: Annest et al. (1983).

Two other patterns, long-term time trends and early childhood age dependent patterns, are applicable to this project. Little is known about age related patterns, but one study in Cincinnati, prior to the project, showed a pattern of blood lead changes during early childhood growth patterns (Figure 2-9).


**Figure 2-9. Expected changes in blood lead during early childhood.**

Source: Adapted from Hasselblad et al. (1980).

Long-term downward trends were documented for child blood lead concentrations during the 1970s and 1980s and have been attributed to decreasing concentrations of lead in food and air. There is little evidence for decreasing concentrations of lead in soil or dust, but sequential measures in these media are rarely made. Data for this project were analyzed for similar trends, and the results are reported in Chapter 3. The QA/QC measures described above and reported in detail in Chapter 3 rule out any possibility of this trend being caused by a measurement artifact such as analytical drift.

There is a question whether that the seasonal cycle is caused by fluctuations in exposure, as opposed to physiological processes that affect the biokinetic distribution of lead within the body. Other studies attribute fluctuations in exposure to changing environmental lead concentrations or changing consumption. During the late summer months, the child may eat food or dust with high lead concentrations or ingest more dust during outdoor play. This project was designed to observe changes in lead concentrations in soil and dust, but not changes in consumption patterns. The observations made on these fluctuations and the interpretation of these observations are reported in Section 3.3.5.

#### 2.3.8.2 Unexplained and Unexpected Sources of Lead

Occasionally, measurements of environmental lead are higher than expected. This section discusses the possibility that such phenomena can be attributed to changes in air concentration alone. Because this study began after the phasedown of lead in gasoline, the air concentrations of lead in these cities had decreased to less than 0.1 $\mu g/m^3$ during the study. The following is a theoretical calculation of the amount of lead that could be transferred to soil or dust from this source alone.

Atmospheric deposition during the study was assumed to be typical for air concentrations that averaged 0.1 $\mu g/m^3$ ($0.1 \times 10^{-6}$ $\mu g/cm^3$). At a deposition rate of 0.2 cm/s, this would accumulate 0.6 $\mu g/cm^2 \cdot$year at the soil surface. Assuming that this lead would be retained in the upper 1 cm of soil surface, 1 $cm^2$ of soil surface equals 1 $cm^3$ of soil, and the annual increment would be 0.6 $\mu g/cm^3$. Because 1 $cm^3$ of soil weighs about 2 g, the annual incremental increase in lead concentration would be 0.3 $\mu g$ Pb/g soil, an insignificant amount in soils that average several hundred $\mu g/g$. The calculation for annual deposition to a surface is

$$1 \times 10^{-7} \frac{\mu g\ Pb}{cm^3} \times 0.2 \frac{cm}{s} \times 3.15 \times 10^7 \frac{s}{year} = 0.6 \frac{\mu g\ Pb}{cm^2\ year} \qquad (2\text{-}2)$$

For the accumulation of dust on hard surfaces, however, the same calculation indicates a potentially greater influence of atmospheric lead. Converting to units of lead loading, and assuming that changes occur over a shorter time period, the 0.6 $\mu g/cm^2 \cdot$year becomes 6,000 $\mu g/m^2 \cdot$year, or 16 $\mu g/m^2 \cdot$day. Therefore, a change of 0.1 $\mu g/m^3$ in air concentration could account for a change of 16 $\mu g$ Pb/$m^2$ per day in the dust lead loading of an up-facing surface. A change of 160 $\mu g/m^2$ over 10 days is in the range of the observed changes in surface dust loading in this project. The impact of atmospheric deposition on exterior dust is discussed in greater detail in Section 3.3.2.

### 2.3.8.3 Movement of Lead in Soil and Dust

In this study, measurements of lead in soil were believed to represent the total amount of lead present in soil, including lead in the crystalline matrix of the parent rock material, in the surfaces of soil particles, in soil moisture, and attached to organic matter. Changes independent of intervention would be expected to occur that would increase lead concentrations as the result of atmospheric deposition, exterior paint chipping and chalking, and human activity such as household waste dumping. Decreasing lead concentrations might occur as a result of leaching downward into the lower soil horizon, or by the reentrainment of surface dust. The downward leaching of lead through the soil profile occurs at a very slow rate. Estimates of a few millimeters per decade are generally considered the most reasonable (Grant et al., 1990). The reentrainment of dust at the soil surface was considered to be in equilibrium with the local environment, such that inputs would equal outputs by this pathway. This is reasonable if there is no flaking or peeling paint within the neighborhood, and no industrial source of fugitive dust in the vicinity of the neighborhood. A limited effort was made to monitor and control the impact of lead-based paint on soil concentrations. Buildings with exterior lead-based paint were stabilized by removal of the chipping and peeling paint, done in a manner to avoid contaminating the soil. There were no attempts to control the introduction of lead to the soil by human activity such as household waste dumping.

In summary, the concentration of lead in soil was expected to be stable throughout the study, but there are a number of reasons why localized soil lead fluctuations might occur.

Lead in household dust is a mixture of that brought into the house from outside and that generated from within the home. Studies have shown that about 85% of the mass of dust comes from outside the home and much of this is apparently brought in on the feet of children and pets (Roberts et al., 1991). In the case where there are no internal sources of lead, such as lead-based paint, the household dust lead concentration is largely a function of the soil concentration in the immediate vicinity of the house. The time delay is unknown, but is believed to be on the order of weeks or months. Thus, changes in soil concentrations would probably appear as changes in household dust concentrations within a few days, and would probably reach equilibrium by 90 days.

## 2.4 REVIEW AND EVALUATION OF INDIVIDUAL STUDY REPORTS

### 2.4.1 Summary of the Boston Study

The Boston study retained 149 of the original 152 children enrolled, although 22 children moved to a new location but were retained in the study. Children with blood lead concentrations below 7 μg/dL or above 24 μg/dL had been excluded from the study and two of the 149 children were dropped from the data analysis when they developed lead poisoning, probably due to exposure to lead-based paint at another location.

Baseline characteristics (age, SES, soil lead, dust lead, drinking water lead, and paint lead) were similar for the three study groups (BOS P, BOS SP, BOS SPI). The preabatement blood lead concentration was higher for BOS P. The proportion of Hispanics was higher in BOS P the BOS SP or BOS SPI, and the proportion of Blacks was lower. There was a larger proportion of male children in BOS P.

Data were analyzed by analysis of covariance (ANCOVA), which showed a significant effect of group assignment (intervention) for both the BOS SP and BOS SPI groups. These results did not change with age, sex, socioeconomic status, or any other variable except race and paint. When the paint variable was added, the effect was diminished; when the race variable was added, the effect became insignificant.

Although designed and conducted to produce rigorous results, the study has several limitations. Participants were chosen to be representative of the population of urban preschool children who are at risk of lead exposure by using the Boston Childhood Lead Poisoning Prevention Program to identify potential participants from neighborhoods with the highest rates of lead poisoning and by using as wide a range of blood lead levels as was practical. Since no study subjects had blood lead levels below 7 μg/dL or in excess of 24 μg/dL at baseline, the study provides no information about the effect of lead contaminated soil abatement for children with these lead levels. Similarly, a different effect might have been found for children who had a greater blood lead contribution from soil, such as in communities with smelters or other stationary sources where soil lead levels are substantially higher than those seen in this study, or where differences in particle size result in differences in bioavailability.

It is possible that the intervention would have been associated with a greater reduction in children's blood lead levels had they been followed for a longer period of time. In addition, all children in the study were exposed to lead contaminated soil prior to enrollment and so we are unable to investigate whether exposure to lead contaminated soil in the first year of life is associated with higher blood lead levels. Lastly, the unit of abatement was the single premises rather than clusters of premises. It is possible that the effect of lead contaminated soil abatement on children's blood lead levels would have been greater had we also removed lead contaminated soil from properties that surrounded Study Group children's premises.

In conclusion, this intervention study suggests that an average 1,856 $\mu g/g$ reduction in soil lead levels results in a 0.8 to 1.6 $\mu g/dL$ reduction in the blood lead levels of urban children with multiple potential sources of exposure to lead.

This study provides information about soil abatement as a secondary prevention strategy, that is the benefit to children already exposed to lead derived, in part, from contaminated soil. It can not be used to estimate the primary prevention effect of soil abatement. Since children's postabatement blood lead levels reflect both recent exposure and body burdens from past exposure, the benefit observed is probably less than the primary prevention benefit, that is the benefit of abating lead contaminated soil before children are exposed to it so as to prevent increases in blood levels and body stores.

### 2.4.2 Summary of the Baltimore Study

The Baltimore study recruited 472 children, of whom 185 completed the study. Of those that completed the study, none were excluded from analysis. The recruited children were from two neighborhoods, originally intended to be a study and a control group. Because soil concentrations were lower than expected, some properties in the study group did not receive soil abatement. The Baltimore report transferred these properties to the control group. In this report, the low soil properties in the study group are treater as a separate group.

Because of logistical problems, there was an extended delay between recruitment and soil abatement that accounted for most of the loss of the participating families from the project. In their report, the Baltimore group applied several statistical models to the two populations to evaluate the potential bias from loss of participating children. These analyses showed the two populations remained virtually identical in demographic, biological and environmental properties.

The Baltimore study was not designed to focus on measurements of the movement of lead through the child's environment. Repeat measurements of soil were on abated properties only, to confirm abatement. There were no measurements of exterior dust, no interior paint stabilization, and no follow-up measurements of house dust. Rather, the study design focused on changes in biological parameters, hand dust and blood lead over an extended period of time.

Including the prestudy screening measurements of hand dust and blood lead in the original cohort of participants, the Baltimore study made six rounds of biological measurements that spanned twenty months. It is unusual to have a data set of this composition and quality. In this integrated report, the baltimore blood lead measurements were the basis for determining the key parameters in the seasonal cycle conversion factor equation discussed in Section 3.3.5.1.

Soil was abated between the third and fourth rounds of biological measurements. The mean soil decrease was 550 $\mu$g/g. At Round 4, the blood lead concentrations were about 0.5 $\mu$g/dL lower in the study group than in the control, or 1 $\mu$g/dL per decrease of 1000 $\mu$g/g in soil, which is comparable to the response observed in the Boston study. By Rounds 5 and 6, the study group blood lead concentrations had returned to their preabatement levels and were in fact higher than the control group.

From the perspective of the Baltimore study alone, it is reasonable to conclude, as the Baltimore report did, that soil abatement has no effect on children's blood lead. But from the perspective of the Boston study, where a blood lead reduction of the same magnitude was found to be persistent when house dust abatement was performed, and from the perspective of the Cincinnati study, where blood lead concentrations were shown to rise and fall in tandem with house dust concentrations, the results of the Baltimore study are consistent with the observation that soil abatement, in conjunction with other environmental interventions, can permanently reduce exposure to lead.

### 2.4.3 Summary of the Cincinnati Study

The Cincinnati study recruited 307 children, including 16 children born to participating families during the study, and an additional 50 children who were recruited after the beginning of the study. In their final report, the Cincinnati group excluded these children who were recruited after the start of the study, plus 31 children who were living in nonrehabilitated housing suspected of having lead-based paint, and four children (in two families) who had become lead-poisoned from other causes. Thus, data for 210 children were analyzed in the Cincinnati report and these same children were included in this integrated report.

The Cincinnati study achieved effective and persistent abatement of soil on the 140 parcels of land scattered throughout the neighborhoods. In CIN SEI, where soil abatement was performed in the first year, the arithmetic mean concentration dropped from 680 $\mu$g/g down to 134 $\mu$g/g. In the two groups where soil abatement occurred in the second year, CIN I-SE-1 and CIN I-SE-2, the soil lead concentration dropped from 262 $\mu$g/g to 125 $\mu$g/g and 724 $\mu$g/g to 233 $\mu$g/g, respectively.

If soil were the only source of lead in the neighborhoods, exterior and interior dust should have responded to the reduction in soil lead concentrations. Exterior dust lead loading decreased following both soil and dust abatement, but returned to preabatement levels within one year. In their report, the Cincinnati group concluded that recontamination of exterior dust began soon after abatement. They observed corresponding changes in house dust, hand lead, and blood lead that paralleled changes in exterior dust. Because blood lead concentrations also decreased in the control area, the Cincinnati group concluded that there is no evidence for the impact of soil and dust abatement on blood lead concentrations. This integrated report concludes, through a more detailed structural equation analysis, that there is a strong relationship between exterior dust and interior dust in this subset of the Cincinnati study where the impact of lead-based paint was minimized. From the perspective of all three studies, this means that when neighborhood and living unit sources of lead are removed, exposure is reduced.

The central hypothesis of the Urban Soil Lead Abatement Demonstration Project is

> *A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*

The formal statement of the Boston hypothesis is

> *A significant reduction (equal to or greater than 1,000 $\mu g/g$) of lead in soil accessible to children will result in a mean decrease of at least 3 $\mu g/dL$ in the blood lead levels of children living in areas with multiple possible sources of lead exposure and a high incidence of lead poisoning.*

The Baltimore hypothesis, stated in the null form, is

> *A significant reduction of lead ($\geq 1,000$ $\mu g/g$) in residential soil accessible to children will not result in a significant decrease (3 to 6 $\mu g/dL$) in their blood lead levels.*

The Cincinnati hypothesis, separated into two parts, is

> *(1) A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*
>
> *(2) Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in blood lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

Secondary hypotheses in the Cincinnati study are

> *(3) A reduction of lead in residential soil accessible to children will result in a decrease in their hand lead levels.*
>
> *(4) Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in hand lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

The array of treatment groups differed considerably among the three studies (Table 2-1). Each treatment group, however, had several features in common. All groups were taken from one to three demographically similar neighborhoods. All groups had some prior evidence of elevated lead exposure, usually a greater than average number of reports of lead poisoning. Each group received the same pattern of treatment: baseline phase for 3 to 18 mo, intervention (except for controls), and followup for 12 to 24 mo.

In each treatment group, even the controls, there was an attempt to minimize the impact of lead-based paint. In Boston, this was done by paint stabilization of both interior and exterior paint. In Baltimore, only exterior paint was stabilized. Therefore, in these two studies, the effects of soil abatement should be evaluated in the context of some intervention for lead-based paint. In Cincinnati, most of the living units had been abated of lead-based paint more than 20 years before the start of the study. Those that had not been abated were measured but not treated prior to the study.

Another difference between the studies was the parallel intervention scheme used in Boston and Baltimore, compared to the staggered scheme used in Cincinnati. In other words, intervention in Boston (and Baltimore) took place at the same time for all treatment groups, and the followup period was of the same duration. But in Cincinnati, the intervention was delayed for one group, CIN I-SE, such that followup varied between 12 and 24 mo.

## 2.5 SUMMARY AND CONCLUSION

### 2.5.1 Summary of Project Description

This project focuses on the exposure environment of the individual child. One measure of short term exposure is the child's blood lead. Two other indicators of exposure are house dust and hand dust. From the perspective of the child's environment, changes in the soil concentration are expected to bring about changes in the house dust concentration, the hand dust concentration, and the blood lead concentration. In each of the three studies, the soil lead concentrations were reduced to approximately 50 $\mu$g/g in the study area, and for most children, there was a measurable reduction of blood lead, although not always statistically significant. When corrected for seasonal and age related cyclic variations on blood lead, the impact was even greater, and the effect was maximized when the rate of movement of dust through the human environment was taken into account. That is, when street dust and house dust were also removed from the environment so that the clean soil represented the major source of lead to the child's environment, the impact of abatement was the greatest.

### 2.5.2 Conclusions

This review of the study designs, analytical procedures and data quality measures has shown no major flaws that would cast doubt on the conclusions of the individual reports. We are now prepared to evaluate the data in a systematic, analytical manner in order to answer the following question: If residential soil is abated will blood lead concentrations decline?

To confirm or reject this soil lead/blood lead hypothesis the reader must pass stepwise through the series of logical arguments described below. Although these statements seem a bit pedantic, each step of the pathway from soil to blood must be scrutinized closely with every detail examined and every possible relationship evaluated. In biogeochemical terms, substances move from one source to another along real, definable pathways. This means that if soil lead is not ingested, either directly or after passing through other sources, then blood lead concentrations cannot respond to changes in soil lead concentrations.

The statements attached to each of these steps give a hint at the conclusions of this report. Data are presented in Chapter 3 that support these statements, followed by statistical inferences in Chapter 4.

1. There is lead in soil.

   Lead was measured in soil in the range of less than 50 $\mu$g/g to more that 18,000 for the combined studies. Each measurement of soil was treated as representing an equal area of soil surface for a given property or soil parcel. If a parcel of 100 $m^2$ had four samples, each with 500 $\mu$g Pb/g soil, then the upper 2 cm of soil on this parcel (about 4,000,000 g) would contain 2,000,000,000 $\mu$g or two kilograms of lead.

2. Lead in soil can move directly onto the child's hand.

   Conceptually, this is difficult to measure, and there is no part of these studies that would confirm this statement. Except for a pica situation, the child is not likely to ingest the same fraction of soil that would be sampled with a 2 cm core. During normal playtime activity, the child would probably get the part of soil that corresponds roughly to playground dust, which is similar to the measurements made of exterior dusts.

3. Lead in soil can also move to other compartments of the child's environment, such as exterior dust.

Evidence for this statement was shown in the Cincinnati study. When lead in soil decreased through abatement, lead in exterior dust also decreased. In the Cincinnati study, however, the relationship between soil and exterior dust was found to be very weak, giving rise to the next question.

4. There are sources of lead other than soil that contribute to exterior dust.

Because the changes in lead in soil do not account for all of the changes in exterior dust, it is reasonable to conclude from the Cincinnati study that there are other sources for lead in exterior dust. In Cincinnati, the soil parcels were not on the individual properties of the participating families, as was the case in Boston and Baltimore. There are no measurements of exterior dust in the Boston or Baltimore studies to confirm or reject the conjecture that exterior dust on the residential study site is more closely linked to lead in soil.

5. Lead in exterior dust can move directly onto the child's hand.

There is no portion of these studies that directly measures this effect. Baltimore reported that the lead loading on hands increased during the summer months, by inference due to the increased playtime outside. During the interviews with the family, questions were asked in all three studies about the activity patterns of the children, including the amount of time spent outside, but none of the studies attempted to determine the play activities immediately before the hand wipe sample was taken.

6. Lead in exterior dust can also move into other components of the child's environment, such as interior dust.

In the Cincinnati study, when exterior dust lead concentrations changed, interior dust lead concentrations also changed. This was especially obvious when the exterior dust sample closest to the residence was compared to the interior floor dust sample taken just inside the entryway door.

A living unit with 130 $m^2$ of floor space (1,400 $ft^2$) and 1,000 $\mu g$ $Pb/m^2$ (a relatively high value from tables in Section 3.3) would have 130,000 $\mu g$ of lead, or less than 1% of the lead available from soil in paragraph 1 above. Additional lead would be in rugs and upholstered furniture.

7. There are sources of lead other than exterior dust that contribute to interior dust.

Taken individually, none of the studies decisively demonstrated this effect. The most obvious source of lead inside the home is lead-based paint, which was common in the Boston and Baltimore studies but excluded from the Cincinnati study. Because neither Boston nor Baltimore measured exterior dust, measurements of interior dust in these studies cannot easily be broken down into contributions from lead-based paint and from exterior dust. However, structural equation analyses on the Boston study showed a string influence of both interior and exterior lead-based paint on interior dust.

8. Lead in interior dust can move directly onto the child's hand.

   In most cased, when interior dust changed, hand dust changed. Because hand dust lead is only a measure of the amount of lead on the hand, not the concentration nor the amount of dust, it is difficult to make a quantitative estimate of this pathway. It is not likely that the amount of dust on the hand is strictly a function of the amount of dust on the playing surface, as there is probably an equilibrium effect where some dust falls off after time. There is no aspect of these studies that could measure this interesting problem.

9. Lead in interior dust can also move into other components of the child's environment, such as food.

   This pathway was not investigated by any of the three studies. Measurements of lead in food before and after kitchen preparation would be required. Conceptually, this lead and other routes such as the direct mouthing activities on toys, furniture, and window sills is included in the measurement of interior dust when the assumption is made that a child ingests about 100 mg of dust per day by all routes and through all activity patterns.

10. There are sources of lead other than dust that contribute to the child's lead exposure.

    In this project, lead was measured in drinking water once or twice during each study. Ambient levels of lead in air were assumed, as were national averages of lead in food. Ethnic food preferences and individual use of cosmetics or other lead containing products were not investigated.

# 3. PROJECT RESULTS

## 3.1 DATA QUALITY

The participating cities recognized the need for standardizing the sampling and analytical protocols so that data from each study could be compared at the end of the project. This was ultimately accomplished for soil and dust by measuring the analytical difference between each the three labs. But before this was possible, common standards needed to be prepared and a program for assuring data quality had to be put into place. A three step program was agreed to that involved: (1) a round robin calibration study of soil samples to measure differences between laboratories and differences between analytical methods and instrumentation, (2) a double blind audit system for soil and dust to monitor the performance of each laboratory during the project, and (3) a second round robin calibration study to determine the arithmetic correction factor that would allow the conversion of dust and soil data to a common project basis. This program would ensure that analyses performed by each of the three participating laboratories would be internally accurate and externally consistent with similar analyses by other research laboratories.

### 3.1.1 Round Robin I: Common Standards and Analytical Methods

At the beginning of this project, the proposed methods for soil and dust analysis were reviewed by the Scientific Coordinating Panel. The preferred method, hot nitric acid digestion followed by atomic absorption spectroscopy (AAS), was time consuming and expensive). The number of samples was expected to exceed 75,000 per project, so more rapid and less expensive methods were evaluated. Laboratory scale X-ray fluorescence (XRF) spectroscopy and inductively coupled plasma (ICP) emission spectroscopy were proposed, and a cold nitric acid extraction method was also considered. The first round robin intercalibration study was organized, as described in Chapter 2. In summary, the test conditions were that each laboratory would be provided with instructions for preparing the samples (drying, sieving, and chemical extraction) but would use their own internal standards and instrumental settings. They would have access to a set of external standards (from U.S. Department of Agriculture) with known values from which they could make corrections if necessary.

Each of the three study laboratories sent aliquots of 10 samples to the other two participating laboratories and to two external laboratories. One of the samples from Cincinnati was a street dust sample with a lead concentration in excess of 15,000 $\mu g/g$. The other 29 samples were soils. The samples were subdivided by sieving during preparation to a "total" and "fine" fraction. Thus there were 30 samples, each with two size factions analyzed by each of five laboratories using either one or two analytical methods. The analytical and wet chemistry methods used by are shown in Table 3-1, and the results of the analyses appear in Table 3-2.

**TABLE 3-1. WET CHEMISTRY AND INSTRUMENTAL METHODS USED FOR THE FIRST INTERCALIBRATION STUDY**

| | Participating Laboratories | | | | |
|---|---|---|---|---|---|
| Method[a] | Boston | Baltimore | Cincinnati | GTRI[b] | USDA[c] |
| Hot $HNO_3$/AAS | | X | X | | |
| Cold $HNO_3$/AAS | | | X | | X |
| Hot $HNO_3$/ICP | | X | | | |
| XRF | X | | | X | |

[a]$HNO_3$ = Nitric acid; AAS = Atomic absorption spectroscopy; ICP = Inductively coupled plasma emission spectroscopy; XRF = X-ray fluorescence.
[b]GTRI = Georgia Tech Research Institute.
[c]USDA = U.S. Department of Agriculture.

The cold nitric acid extraction method was found to be essentially equivalent to the hot nitric acid extraction method for soils with lead concentrations up to 8,000 $\mu g/g$ (Figure 3-1) for the samples analyzed in this study. The AAS method used by Cincinnati and Baltimore was also equivalent (Figure 3-2), showing a high degree of comparability between these two laboratories under these test conditions.

The interlaboratory comparison of X-ray fluorescence (XRF) between the Boston and Georgia Tech Research Institute (GTRI) Laboratories showed the method was acceptable, although not fully linear above 5,000 $\mu g/g$. There were no soil standards available above 2,000 $\mu g/g$ so the analysts had some difficulty calibrating their XRF instruments above this level. The data of Figure 3-3 suggest a systematic difference between the two laboratories

## TABLE 3-2. ANALYTICAL RESULTS OF THE FIRST INTERCALIBRATION STUDY: LEAD CONCENTRATION ($\mu g/g$) IN THE TOTAL AND FINE FRACTIONS OF 10 SOILS FROM EACH STUDY

| Sample Fraction[c] | Boston XRF | Baltimore Hot $HNO_3$ AAS | Baltimore Hot $HNO_3$ ICP | Cincinnati Hot $HNO_3$ AAS | Cincinnati Cold $HNO_3$ AAS | GTRI[a] XRF | USDA[b] Cold $HNO_3$ AAS |
|---|---|---|---|---|---|---|---|
| 1T | 1,200 | 1,418 | 1,324 | 1,552 | 1,215 | 1,174 | 1,338 |
| 2T | 1,750 | 2,893 | 2,544 | 2,868 | 2,211 | 1,912 | 2,695 |
| 3T | 400 | 492 | 389 | 387 | 466 | 400 | 417 |
| 4T | 550 | 619 | 462 | 423 | 415 | 500 | 464 |
| 5T | 1,100 | 1,058 | 882 | 964 | 854 | 980 | 988 |
| 6T | 1,450 | 2,323 | 1,955 | 1,876 | 1,722 | 1,524 | 1,808 |
| 7T | 1,000 | 1,359 | 1,098 | 1,383 | 990 | 651 | 1,473 |
| 8T | 500 | 683 | 535 | 491 | 725 | 400 | 726 |
| 9T | 550 | 608 | 485 | 455 | 417 | 261 | 605 |
| 10T | 1,450 | 1,649 | 1,330 | 1,679 | 1,228 | 1,660 | 1,764 |
| 11T | 250 | 484 | 365 | 316 | 348 | 180 | 304 |
| 12T | 800 | 1,069 | 878 | 1,850 | 1,103 | 900 | 1,944 |
| 13T | 100 | | 53 | 63 | 45 | 100 | 73 |
| 14T | 700 | 2,200 | 1,701 | 2,068 | 1,713 | 652 | 1,710 |
| 15T | 550 | 1,754 | 1,410 | 747 | 785 | 505 | 825 |
| 16T | 220 | 264 | 200 | 253 | 295 | 187 | 286 |
| 17T | 220 | 126 | 62 | 59 | 58 | 30 | 83 |
| 18T | 75 | 106 | 48 | 74 | 61 | 100 | 111 |
| 19T | 50 | 9 | 7 | 2 | 3 | 20 | 13 |
| 20T | 4,800 | 15,792 | 12,030 | 14,593 | 8,147 | 4,817 | 14,733 |
| 21T | 500 | 496 | 372 | 387 | 378 | 383 | |
| 22T | 950 | 850 | 698 | 837 | 739 | 717 | 1,120 |
| 23T | 1,700 | 1,559 | 1,298 | 1,567 | 1,368 | 1,390 | 1,761 |
| 24T | 2,400 | 2,260 | 1,880 | 2,284 | 2,003 | 2,021 | 2,561 |
| 26T | 2,800 | 2,484 | 2,119 | 2,754 | 2,401 | 2,331 | 2,472 |
| 27T | 3,800 | 3,846 | 3,440 | 4,337 | 3,835 | 3,500 | 4,983 |
| 28T | 5,200 | 5,092 | 4,667 | 5,454 | 4,747 | 4,460 | 3,184 |
| 29T | 4,000 | 5,097 | 4,510 | 5,586 | 4,700 | 3,280 | 6,473 |
| 30T | 6,500 | 7,995 | 6,560 | 8,467 | 7,502 | 4,704 | 10,042 |
| 1F | 1,500 | 1,545 | 1,421 | 1,560 | 1,404 | 1,223 | 1,569 |
| 2F | 2,650 | 3,540 | 2,921 | 3,335 | 3,127 | 2,263 | 3,273 |
| 3F | 500 | 625 | 507 | 478 | 508 | 440 | 515 |
| 4F | 1,600 | 1,814 | 1,554 | 1,678 | 1,595 | 1234 | 1,824 |
| 5F | 1,700 | 1,793 | 1,475 | 1,689 | 1,971 | 1,290 | 1,683 |
| 6F | 2,400 | 3,137 | 2,387 | 2,835 | 2,009 | 2,134 | 2,682 |
| 7F | 1,200 | 1,344 | 1,105 | 1,306 | 1,184 | 815 | 1,297 |
| 8F | 600 | 723 | 598 | 595 | 298 | 490 | 672 |
| 9F | 650 | 686 | 558 | 593 | 601 | 375 | 630 |
| 10F | 2,200 | 2,398 | 1,946 | 1,808 | 1,116 | 1,980 | |
| 11F | 220 | 356 | 244 | 267 | 277 | 180 | 280 |
| 12F | 1,800 | 2,707 | 2,220 | 2,683 | 2,683 | 1,680 | 2,610 |
| 13F | 100 | 96 | 68 | 68 | 64 | 100 | 89 |
| 14F | 800 | 100 | 779 | 926 | 818 | 693 | 895 |
| 15F | 620 | 796 | 616 | 635 | 642 | 600 | 664 |
| 16F | 300 | 3,200 | 236 | 237 | 239 | 236 | 242 |
| 17F | 100 | 118 | 73 | 73 | 66 | 100 | 80 |
| 18F | 100 | 142 | 85 | 91 | 87 | 100 | 92 |
| 19F | 50 | | 10 | 3 | 2 | 30 | 20 |
| 20F | 5,100 | 7,866 | 6,000 | 8,109 | 7,432 | 4,780 | 8,451 |
| 21F | 550 | 606 | 506 | 480 | 467 | 505 | 470 |
| 22F | 1,100 | 1,118 | 916 | 1,069 | 944 | 980 | 904 |

**TABLE 3-2 (cont'd). ANALYTICAL RESULTS OF THE FIRST INTERCALIBRATION STUDY: LEAD CONCENTRATION ($\mu g/g$) IN THE TOTAL AND FINE FRACTIONS OF 10 SOILS FROM EACH STUDY**

| | Boston | Baltimore | | Cincinnati | | GTRI[a] | USDA[b] |
|---|---|---|---|---|---|---|---|
| Sample Fraction[c] | XRF | Hot $HNO_3$ AAS | Hot $HNO_3$ ICP | Hot $HNO_3$ AAS | Cold $HNO_3$ AAS | XRF | Cold $HNO_3$ AAS |
| 23F | 1,700 | 1,679 | 1,424 | 1,710 | 1,431 | 1,320 | 1,640 |
| 24F | 2,200 | 2,331 | 2,014 | 2,328 | 2,010 | 1,940 | |
| 25F | 2,200 | 2,372 | 2,000 | 1,665 | 2,089 | 2,005 | 2,492 |
| 26F | 2,800 | 2,899 | 2,402 | 2,946 | 2,568 | 2,249 | 3,156 |
| 27F | 4,000 | 4,833 | 3,969 | 4,531 | 4,130 | 3,739 | 4,979 |
| 28F | 3,100 | 3,087 | 2,616 | 3,073 | 2,720 | 2,445 | 6,194 |
| 29F | 4,500 | 5,896 | 4,717 | 5,606 | 4,869 | 4,240 | 6,680 |
| 30F | 8,000 | 8,555 | 7,443 | 8,679 | 7,789 | 6,015 | 9,754 |

[a]GTRI = Georgia Tech Research Institute.
[b]USDA = U.S. Department of Agriculture.
[c]T = Total fraction, F = Fine fraction.

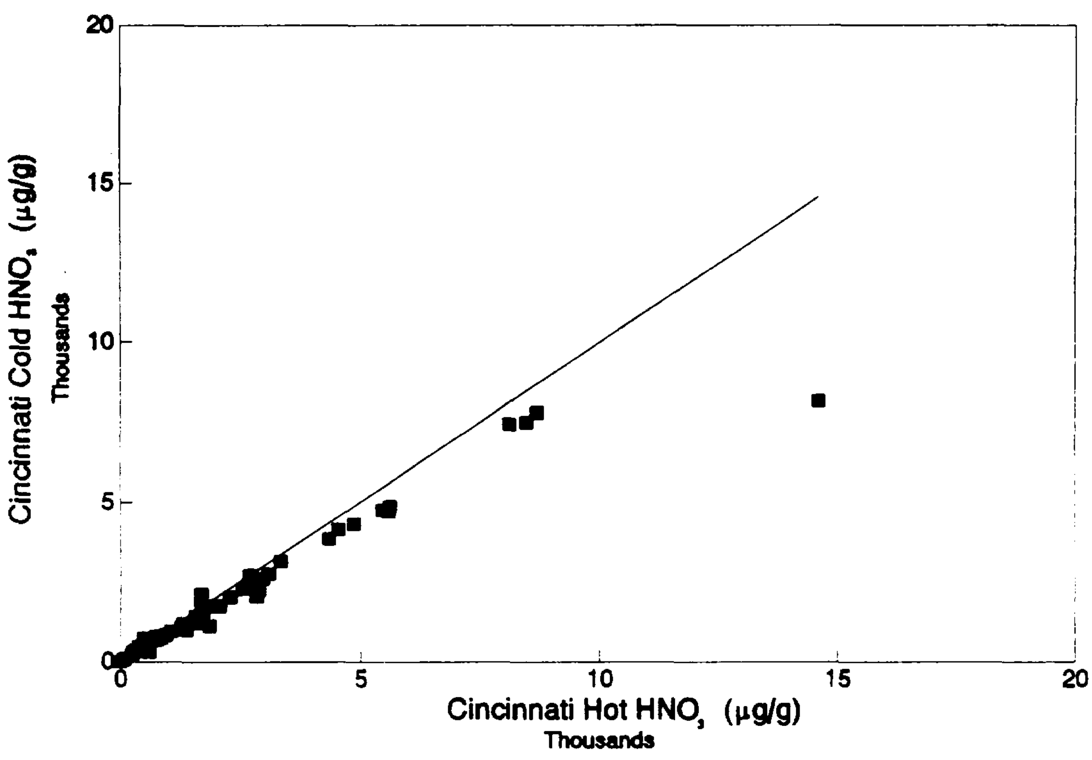


**Figure 3-1. Comparison of uncorrected data for two wet chemistry methods of soil analysis showing the comparability of hot and cold nitric acid for the Cincinnati laboratory. The straight line indicates a slope of 1.**

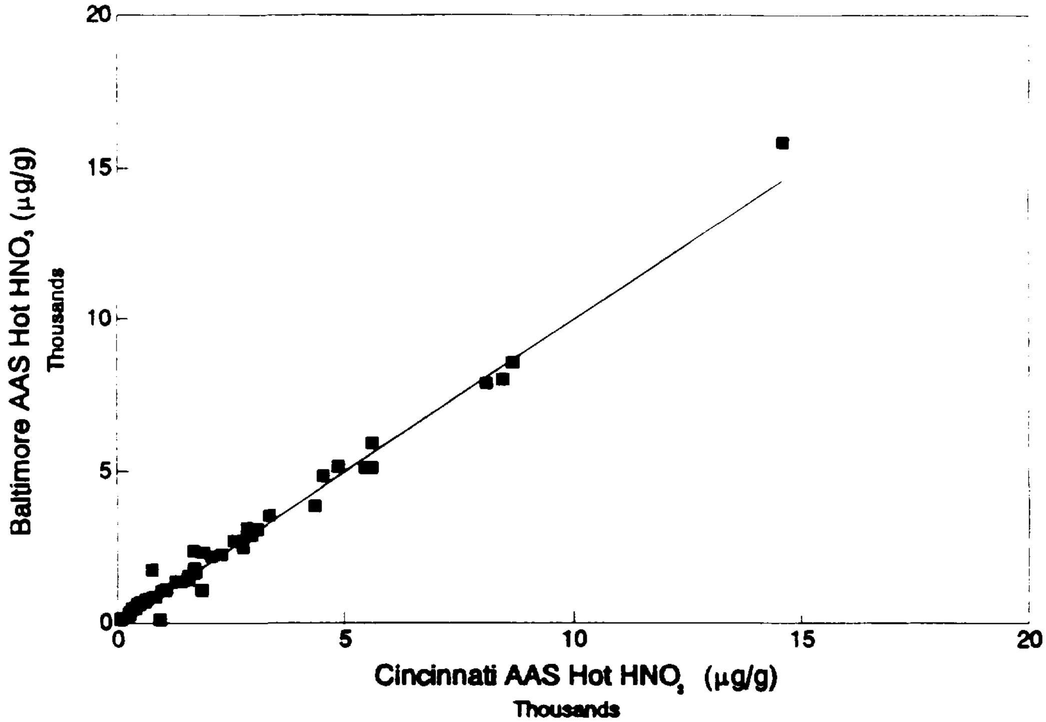


**Figure 3-2. Comparison of uncorrected data for atomic absorption spectroscopic analysis by two laboratories (Baltimore and Cincinnati) using the hot nitric acid method of soil analysis. The straight line indicates a slope of 1.**

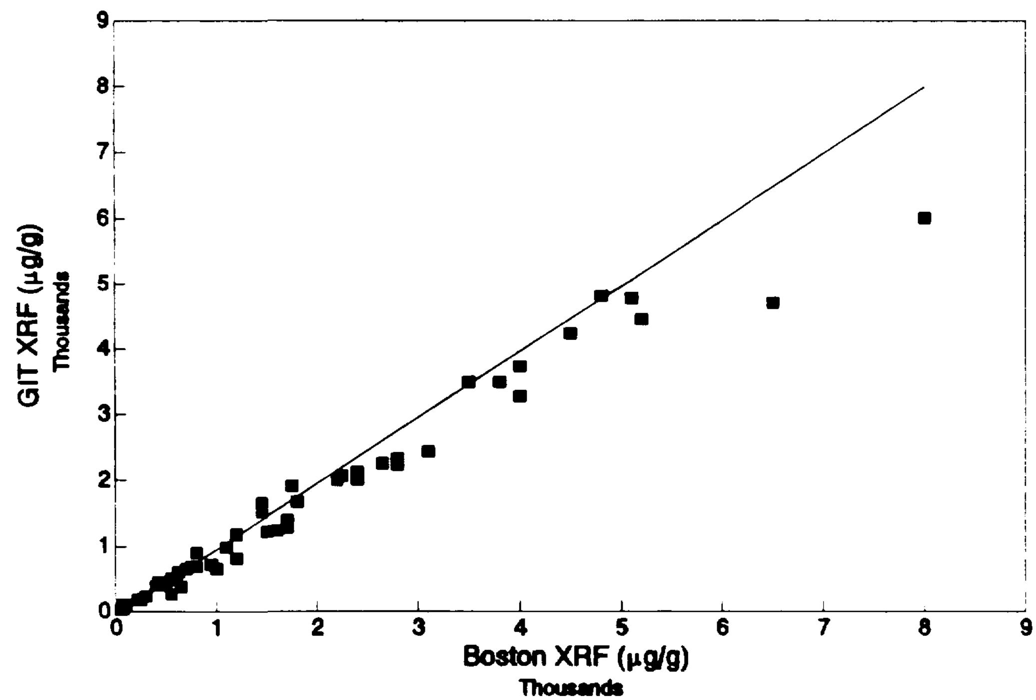


**Figure 3-3. Interlaboratory comparison of uncorrected data for the X-ray fluorescence method of soil analysis showing the comparability of the Boston and Georgia Institute of Technology laboratories. The straight line indicates a slope of 1.**

that could be corrected with a more uniform calibration. Both interlaboratory (Cincinnati and Baltimore in Figure 3-4) and intralaboratory (Baltimore in Figure 3-5) comparisons of AAS versus ICP demonstrated equivalency between these two instrumental methods. These comparisons showed that there is likewise a systematic difference that can be corrected arithmetically.

Finally, the interlaboratory comparison of XRF versus AAS (Boston and Cincinnati in Figure 3-6, and Boston and Baltimore in Figure 3-7) led to the conclusion that of suitable soil standards at higher concentration could be made available, XRF is an acceptable alternative method to AAS for soil analysis.

Based on this study and the awareness that chemical extraction of 75,000 soil samples presented a costly burden on the project both in terms of time and expense, and the value in that nondestructive analysis would preserve the samples for reanalysis, the Scientific Coordinating Panel recommended the use of XRF for soil analysis on the condition that a suitable set of common standards could be prepared for a broader concentration range and that a rigorous audit program be established to ensure continued analytical accuracy.

The Round Robin I calibration exercise also raised the need for a broader scale calibration exercise to determine the arithmetic correction factor for converting the data to a common basis. For routine analyses, two groups, Boston and Baltimore, elected to use XRF for dust analysis also, whereas Cincinnati opted for hot nitric extraction with AAS. During the study, Baltimore recognized problems with analyzing dust by XRF when the sample size was small, less than 100 mg. They reanalyzed the dust samples by AAS and reported both measurements. In Boston, this problem was solved by compositing the floor dust samples for XRF analysis, reporting one floor dust sample per housing unit.

### 3.1.2 Double Blind Audit Program for Soil and Dust

The procedures for the audit program are discussed in Section 2.3.6.1. The results of that program are given in Table 3-3 based on the find biweight distributions in Table 3-4. The preliminary biweight distributions, shown also in Table 3-4, contained no measure of interlaboratory variability because the preliminary analyses were performed by only the EMSL-LV laboratory. Thus they could only be used in the audit program for identifying and

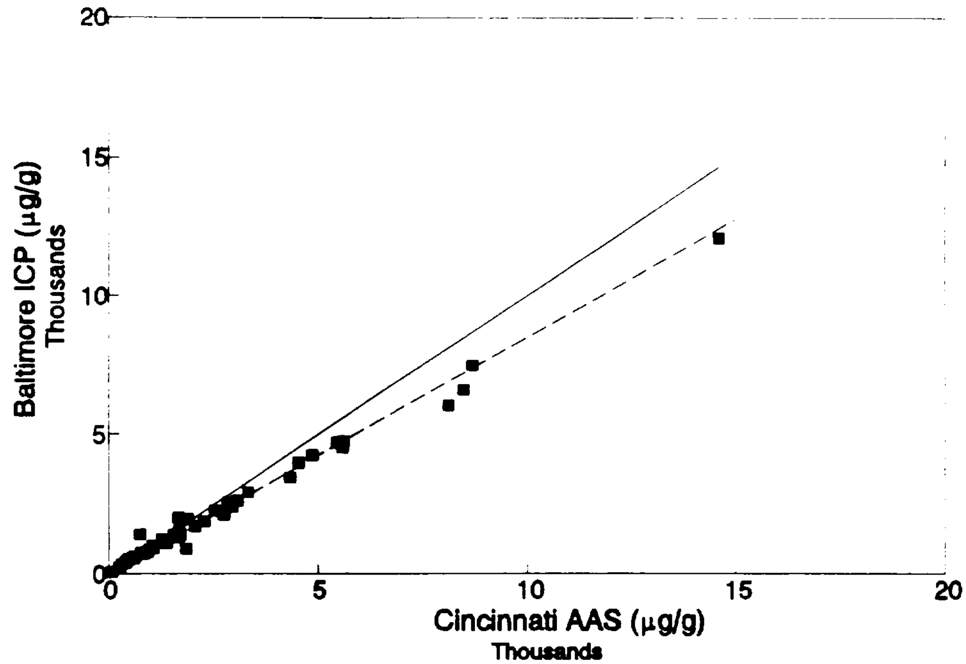


**Figure 3-4. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of inductively coupled plasma emission spectroscopy and atomic absorption spectroscopy for the Baltimore and Cincinnati laboratories. The straight line indicates a slope of 1.**

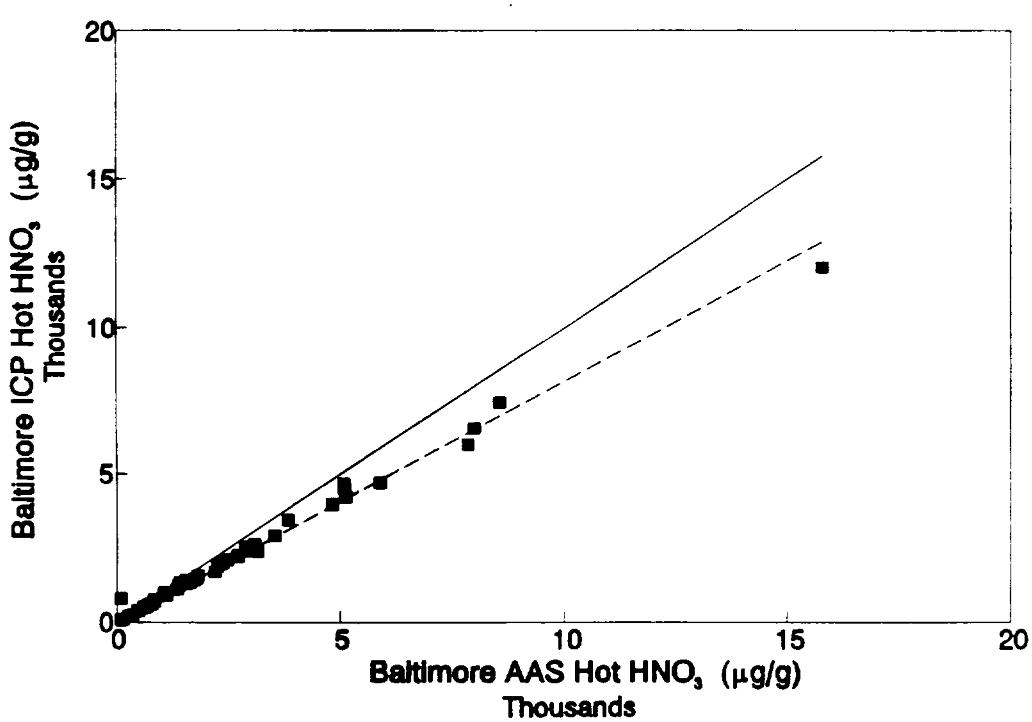


**Figure 3-5. Comparison of uncorrected data for soil analysis showing the comparability of inductively coupled plasma emission spectroscopy and atomic absorption spectroscopy within the Baltimore laboratory. The straight line indicates a slope of 1.**

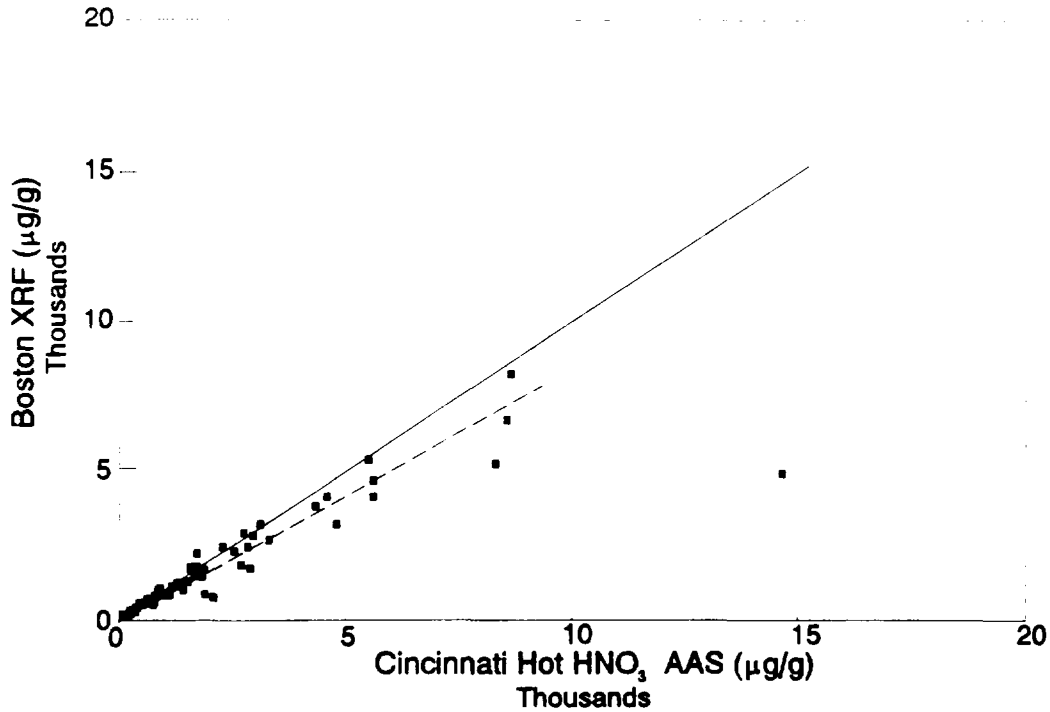


**Figure 3-6. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of X-ray fluorescence and atomic absorption spectroscopy for the Cincinnati and Boston laboratories. The straight line indicates a slope of 1.**

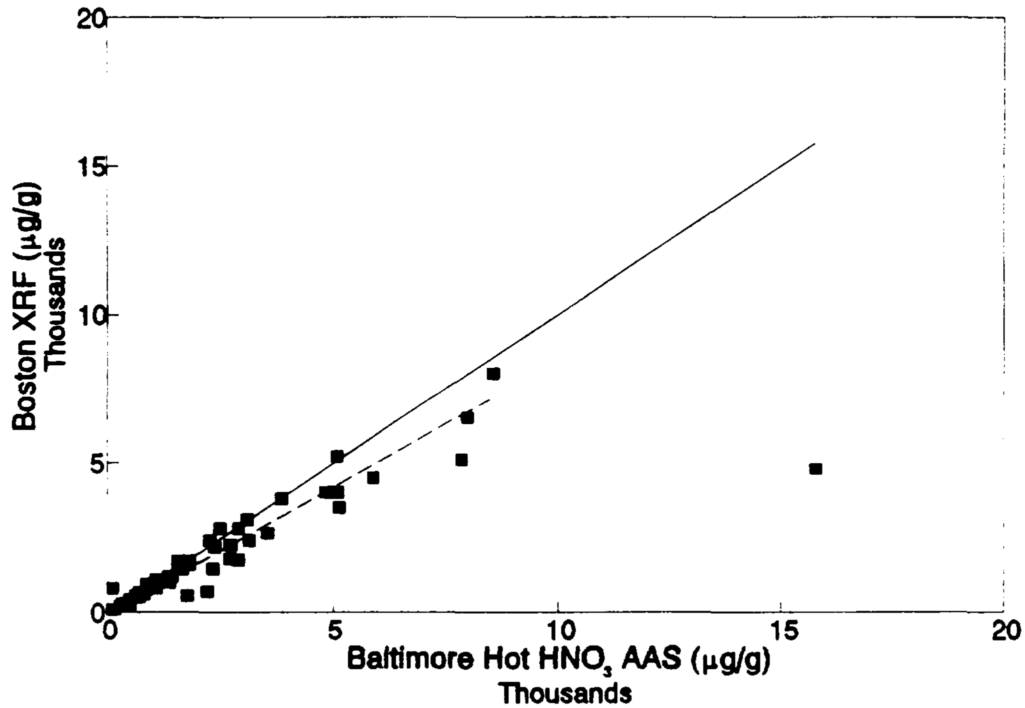


**Figure 3-7. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of X-ray fluorescence and atomic absorption spectroscopy for the Baltimore and Boston laboratories. The straight line indicates a slope of 1.**

TABLE 3-3. SOIL AND DUST AUDIT PROGRAM RESULTS

| Study/Audit Sample | Number of Samples | Mean | Range | Percent Within Final Biweight Distribution |
|---|---|---|---|---|
| BOSTON DUST | | | | |
| BAL 03 | N/A[a] | 1,232 | 980-1,441 | 92 |
| CIN 01 | N/A | 2,671 | 2,075-3,228 | 100 |
| CIN 02 | N/A | 331 | 115-461 | 65 |
| BOSTON SOIL | | | | |
| BOS M | N/A | 6,786 | 6,015-7,549 | 100 |
| BAL H | N/A | 1,044 | 747-1,244 | 73 |
| CIN L | N/A | 399 | 207-570 | 61 |
| CIN H | N/A | 14,074 | 11,407-16,592 | 50 |
| BALTIMORE DUST (XRF) | | | | |
| BAL 02 | 8 | 218 | 159-281 | 100 |
| CIN 01 | 10 | 3,280 | 800-3,660 | 90 |
| BOS 01 | 10 | 14,444 | 14,080-14,920 | 100 |
| BALTIMORE SOIL | | | | |
| BOS M | 15 | 5,046 | 4,800-4,200 | 100 |
| BAL H | 15 | 838 | 433-916 | 60 |
| CIN L | 15 | 286 | 266-307 | 100 |
| CIN H | 15 | 11,290 | 10,100-12,500 | 53 |

| Study | Number of Samples | Mean | STD DEV | Percent Within Final Biweight Distribution |
|---|---|---|---|---|
| CINCINNATI DUST (AAS) | | | | |
| BAL 03 | 34 | 1,727 | 275 | N/A |
| BOS 01 | 35 | 24,104 | 2,337 | N/A |
| CIN 01 | 38 | 2,683 | 225 | 100 |
| CIN 02 | 26 | 259 | 44 | 100 |
| CINCINNATI SOIL | | | | |
| BOS M | 32 | 6,654 | 268 | 100 |
| BAL H | 49 | 1,016 | 40 | 100 |
| CIN L | 130 | 301 | 11 | 100 |
| CIN H | 31 | 14,890 | 635 | N/A |

[a]N/A = Not available.

TABLE 3-4. PRELIMINARY AND FINAL BIWEIGHT DISTRIBUTIONS FOR SOIL AND DUST AUDIT PROGRAM

| Sample Type | Audit Sample | Preliminary Values | | | Final Values | | |
|---|---|---|---|---|---|---|---|
| | | Mean | Low | High | Mean | Low | High |
| Dust | BAL01 | 78 | 58 | 99 | 84 | 4 | 163 |
| Dust | BAL02 | 331 | 288 | 374 | 309 | 138 | 480 |
| Dust | BAL03 | 1.480 | 1.346 | 1,613 | 1.438 | 1.091 | 1.786 |
| Dust | CIN01 | 2.851 | 2.660 | 3.042 | 2.617 | 1.422 | 3.812 |
| Dust | CIN02 | 252 | 216 | 288 | 233 | 93 | 372 |
| Soil | BOS L | 3,131 | 2.858 | 3,405 | 3,101 | 2,283 | 3.919 |
| Soil | BOS M | 6,090 | 5.748 | 6,431 | 6.219 | 4.742 | 7.696 |
| Soil | BOS H | 14,483 | 13.071 | 15,895 | 13,369 | 11,980 | 14,754 |
| Soil | BAL L | 639 | 555 | 724 | 626 | 468 | 783 |
| Soil | BAL H | 923 | 850 | 997 | 1,017 | 847 | 1,187 |
| Soil | CIN L | 303 | 284 | 322 | 315 | 204 | 426 |
| Soil | CIN H | 13,585 | 12,872 | 14,297 | 12,729 | 11,361 | 14,096 |
| Soil | REF5 | | | | 413 | 258 | 568 |
| Soil | REF6 | | | | 936 | 738 | 1,134 |
| Soil | REF7 | | | | 1,042 | 758 | 1,326 |
| Soil | REF8 | | | | 2,354 | 1,950 | 2,759 |
| Soil | REF9 | | | | 3,913 | 2,943 | 4,888 |
| Soil | REF10 | | | | 735 | 615 | 854 |

flagging batches of soil samples that might need to be reanalyzed pending the determination of the final biweight distributions.

As the audit program progressed, two patterns emerged. In some cases, laboratories were systematically low or high, and this was not of major concern, as these discrepancies could be resolved by a more detailed intercalibration exercise and arithmetic correction. The Cincinnati group elected to make a midcourse change in instrumental parameters that reduced this difference, as described in the Cincinnati report. In other cases, the measured audit sample was sporadically high or low, in which case the laboratories investigated the problem and resolved it. Most of these discrepancies occurred for dust samples where the sample size for XRF analysis was below 200 mg. Here, the Baltimore group elected to analyze by AAS and the Boston group composited the floor samples from several rooms in a single residence to obtain a larger sample size. The Boston group also found, but did not report in detail, that a calibration curve for XRF analysis using standards that were also less than 200 mg would provide a suitable correction to the original data.

Batch analyses with audit discrepancies that could not be resolved by one of these two means were retained in the data set but flagged. This was necessary during the study because the final values for the upper and lower acceptance limits could not be determined until the end of the study.

### 3.1.3 Round Robin II: Biweight Distribution and Final Interlaboratory Calibration

The nine soil and five dust samples that were used for external standards and audit samples were reanalyzed in a more detailed round robin exercise near the end of the project, as described in Section 2.3.6.1. The purpose of this exercise was to determine the correction factor for mathematically converting the soil and dust data from each study to a common basis and to revise the biweight distribution values for the audit samples to reflect the multilaboratory variance and systematic differences between laboratories. Additional analyses by AAS were performed by Baltimore and Cincinnati for soil and dust, even though only dust was analyzed by AAS during the study. Boston and Las Vegas analyzed the samples by ICP for the purposes of obtaining a broader perspective on the application of this method. The data from this exercise are in Table 3-5. They are the basis for determining the consensus values and correction factors that appear in Table 3-6.

The data evaluation subcommittee of the Scientific Coordinating Panel was appointed to determine the consensus values and methods of statistical interpretation of the intercalibration results. Several methods were discussed in great detail. Tests were made for outliers using the method of Barnett and Lewis (1984), and none were found. The data were of good quality and were highly linear. The $r^2$ values ranged from 0.997 to 0.999 using a consensus based on the simple arithmetic means of the reported values. The subcommittee chose to

**TABLE 3-5. RESULTS OF THE FINAL INTERCALIBRATION STUDY**

| | XRF | | | | | AAS | | ICP | |
|---|---|---|---|---|---|---|---|---|---|
| Sample | BOSK | BOSX | BAL | CIN | LV | BAL | CIN | BOS | LV |
| DUST1 | 120 | | 121 | 92 | 78 | 15 | 66 | 94 | 72 |
| DUST2 | 320 | | 482 | 329 | 288 | 201 | 236 | 284 | 307 |
| DUST3 | 1,430 | | 1,686 | 1,307 | 1,288 | 1,363 | 1,581 | 1,428 | 1,346 |
| DUST4 | 2,000 | | 3,771 | 2,924 | 2,456 | 2,335 | 2,451 | 2,109 | 2,296 |
| DUST5 | 280 | | 267 | 233 | 212 | 150 | 273 | 244 | 191 |
| SOIL1 | 450 | 510 | 388 | 441 | 310 | 383 | 452 | 401 | 379 |
| SOIL2 | 900 | 910 | 808 | 1,033 | 833 | 1,001 | 1,013 | 850 | 912 |
| SOIL3 | 1,050 | 1,100 | 961 | 1,080 | 923 | 1,100 | 1,120 | 972 | 1,006 |
| SOIL4 | 2,200 | 2,300 | 2,100 | 2,555 | 2,264 | 2,468 | 2,502 | 2,230 | 2,286 |
| SOIL5 | 3,800 | 4,000 | 3,486 | 4,227 | 3,974 | 4,044 | 4,251 | 3,748 | 3,843 |
| SOIL6 | 710 | 770 | 640 | 789 | 611 | 741 | 798 | 699 | 660 |
| SOIL7 | 650 | 930 | 559 | 675 | 532 | 567 | 650 | 597 | 626 |
| SOIL8 | 950 | 930 | 896 | 1,036 | 798 | 1,032 | 1,067 | 944 | 998 |
| SOIL9 | 2,800 | 2,900 | 2,514 | 3,126 | 2,972 | 3,401 | 3,263 | 3,148 | 3,158 |
| SOIL10 | 5,600 | 5,300 | 5,200 | 6,493 | 5,956 | 6,861 | 6,937 | 5,932 | 6,360 |
| SOIL11 | 12,500 | 13,000 | 11,000 | 15,963 | 15,984 | 13,175 | 13,955 | 12,652 | 12,608 |
| SOIL12 | 310 | 290 | 283 | 305 | 286 | 321 | 379 | 300 | 294 |
| SOIL13 | 12,000 | 12,000 | 10,500 | 14,156 | 13,530 | 13,000 | 13,195 | 13,167 | 11,440 |
| SOIL14 | 810 | 850 | 793 | 929 | 763 | 875 | 986 | 907 | 900 |
| SOIL15 | 1,450 | 1,600 | 1,400 | 1,705 | 1,509 | 1,731 | 1,766 | 1,631 | 1,650 |

explore alternatives to the arithmetic mean and eventually settled on a multiplicative model weighted for within-laboratory variance. The model was run with GLIM statistical software, Version 3.77, Update 2, and gave consensus values and correction factors shown in Table 3-6. Although great care was taken to evaluate several alternatives to simple regression, the consensus values produced by the GLIM procedure differed only slightly from those of a simple linear regression. The correction factors on Table 3-6 were used by the three studies to convert their soil and dust data to a common project basis. A plot of the

TABLE 3-6. CONSENSUS VALUES AND CORRECTION FACTORS FROM THE FINAL INTERCALIBRATION PROGRAM

| | XRF | AAS | ICP |
|---|---|---|---|
| | Interlaboratory Consensus Values for Dust | | |
| Sample | | | |
| DUST1 | 92.8 | 54.2 | 81.7 |
| DUST2 | 342.7 | 221.9 | 283.4 |
| DUST3 | 1,319.0 | 1,492.2 | 1,362.3 |
| DUST4 | 2,943.4 | 2,378.1 | 2,133.4 |
| DUST5 | 228.3 | 232.4 | 206.2 |
| | Interlaboratory Correction Factors | | |
| Study | | | |
| BOS | 1.1527 | | 1.0707 |
| BAL | 0.7803 | 1.0416 | |
| CIN | 1.0074 | 0.9616 | |
| | Interlaboratory Consensus Values for Soil | | |
| Sample | | | |
| SOIL1 | 460.2 | 430.5 | 426.6 |
| SOIL2 | 960.7 | 1,002.1 | 909.6 |
| SOIL3 | 1,140.5 | 1,106.2 | 1,018.8 |
| SOIL4 | 2,493.5 | 2,474.2 | 2,342.1 |
| SOIL5 | 4,139.3 | 4,164.1 | 3,706.1 |
| SOIL6 | 761.0 | 776.9 | 736.1 |
| SOIL7 | 664.1 | 623.3 | 656.0 |
| SOIL8 | 1,062.3 | 1,049.4 | 1,005.4 |
| SOIL9 | 2,987.8 | 3,272.6 | 3,274.9 |
| SOIL10 | 6,175.2 | 6,863.2 | 6,411.5 |
| SOIL11 | 13,120.7 | 13,645.4 | 13,224.7 |
| SOIL12 | 335.3 | 361.5 | 323.6 |
| SOIL13 | 12,498.5 | 13,041.6 | 13,080.0 |
| SOIL14 | 941.3 | 949.5 | 923.3 |
| SOIL15 | 1,663.2 | 1,744.1 | 1,716.8 |
| | Interlaboratory Correction Factors for Soil | | |
| Study | | | |
| BOS | 1.0370 | | 1.0166 |
| BAL | 1.1909 | 1.0166 | |
| CIN | 0.8698 | 0.9839 | |

dust (Figure 3-8) and soil (Figure 3-9) reported values versus the consensus means derived from the GLIM analysis illustrates the reliability of this method.

### 3.1.4 Disposition of Audit Data

Based on the results of the second intercalibration exercise, a consensus value was determined for each dust and soil sample, biweight distributions were determined for those that had been used in the audit program. This new distribution incorporated interlaboratory variation. When the correction factor is applied to the reported audit samples results, the revised number should lie between the upper and lower boundaries of the biweight distribution. Table 3-3 lists the number and percentage of these audit sample values that fell within these new boundaries. Most of the discrepancies were resolved by the corrective measured taken by the laboratories as described in Section 3.1.2.

When the audit sample values fell outside the boundaries of the final biweight distribution the batches were flagged but not rejected. This decision was made for two reasons. The quality of soil and dust analysis in this project was a step or two above the acceptable standards for research studies involving soil and dust analyses. Furthermore, to attempt to raise this level one more step would have been costly and would have produce little more in terms of scientific information. The simple fact is that if these data had been rejected because of discrepancies with the audit samples, then all previous and subsequent research studies in which a double blind audit program was not used would also have to be rejected. By flagging the batches of data associated with these outlying audit samples the groups and other users of the data could attempt to determine, on a one-to-one basis, the best explanation for the apparent discrepancy. The options could be to exclude these data from their statistical analysis, reanalyze the samples, or use the original data based on the assumption that the data are correct.

### 3.1.5 Database Quality

Each study maintained rigorous standards for database quality. These included double entry, 100% visual confirmation, and standard procedures for detecting outliers. In spite of these measures, some errors were found, confirmed, and corrected prior to use in this report. None of these errors would have impacted the conclusions drawn by the individual study.

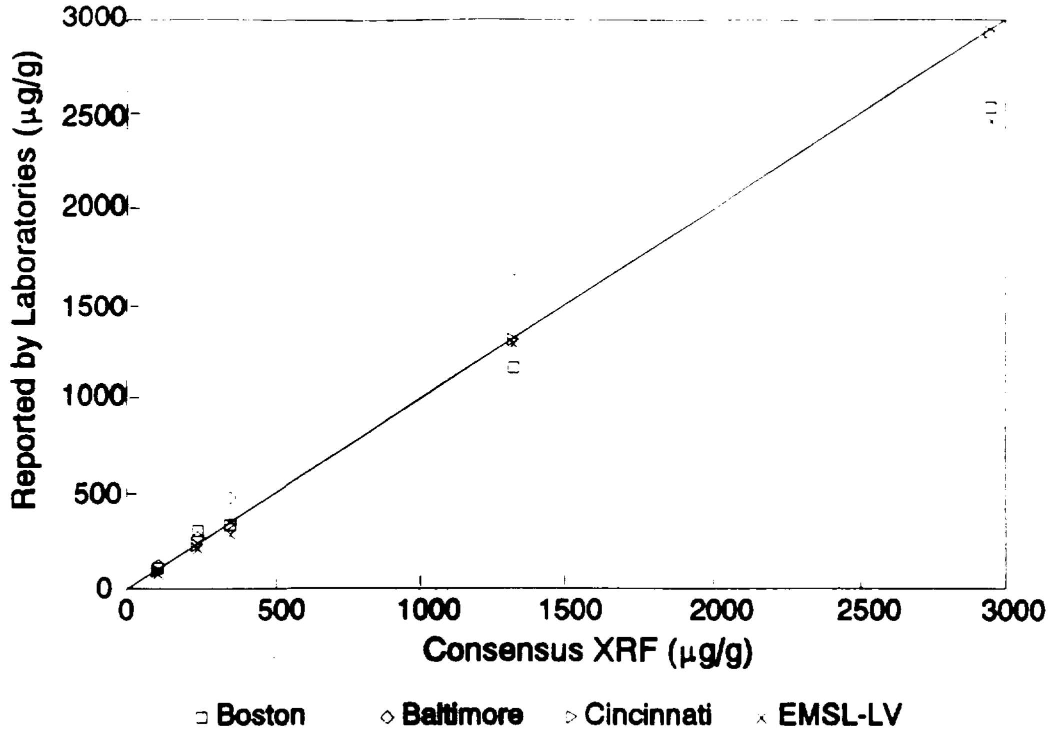


**Figure 3-8. Departures from consensus dust values for each of the three studies.**

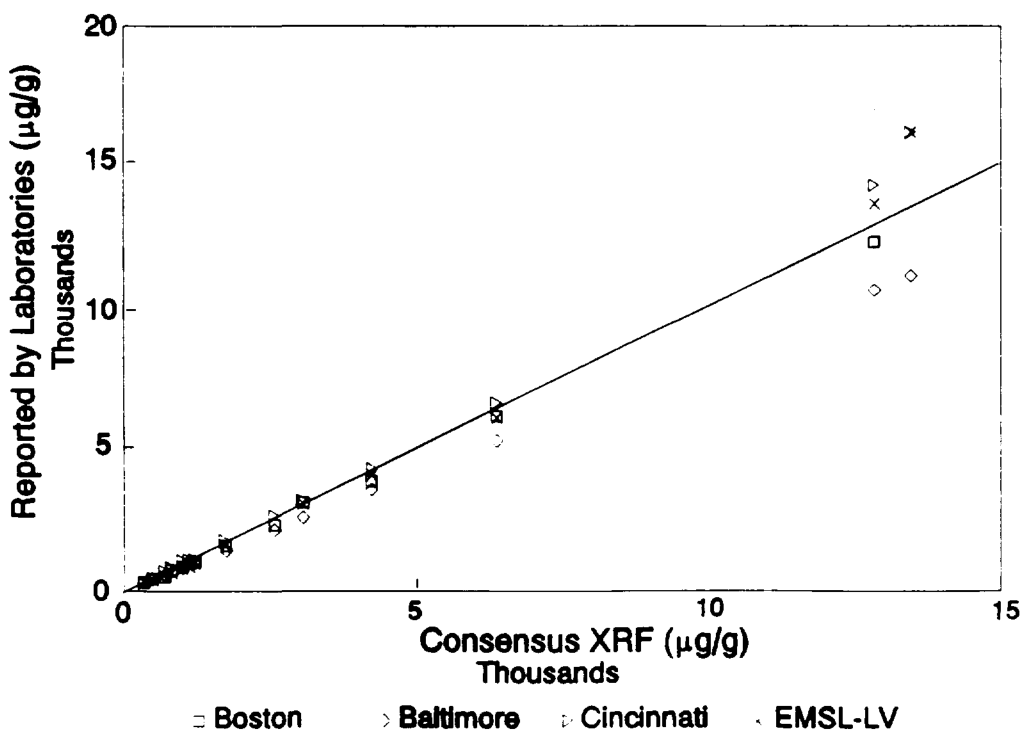


**Figure 3-9. Departures from consensus soil values for each of the three studies.**

## 3.2 OVERVIEW OF PROJECT DATA

### 3.2.1 Description of the Data

#### 3.2.1.1 Types of Data

The analytical data used in this report consist of measurements of soil, exterior dust (sometimes referred to as street dust), interior dust (house dust), hand dust, blood lead, exterior paint, interior paint, and drinking water. The age and sex of the child and the date of collection are also included in these analyses.

#### 3.2.1.2 Data Collection Patterns

Each study produced the same or similar information about the occurrence of lead in the environment. The data sets among the studies are not identical, however, in that they differed in the timing of the collection relative to intervention, the spatial distribution of the sampling points relative to the expected exposure to the child, and the manner in which the data were reduced to a central tendency.

Data were collected in rounds. That is, during a specific period of time, samples were taken of soil, dust, etc., for a specific objective, such as establishing the concentration of lead prior to intervention. Usually a round lasted for several weeks, perhaps 3 to 4 mo. Rounds are not contiguous, in that there were gaps during which no samples were taken. It is easy to see that it may be important to know when a sample was taken during a round, especially following intervention, in order to evaluate the impact on exposure. Consider the pathway from soil ⇒ street dust ⇒ house dust ⇒ hand lead ⇒ blood lead. One would expect, if soil alone (not house dust) were abated and the exposure were mainly through house dust, there would be a lag between abatement and response, and the impact of intervention might become greater with increasing time. Conversely, the impact of intervention might be reduced with time if there were recontamination, as would be expected if house dust were abated but soil were not.

#### 3.2.1.3 Data Linkages

Data linkages are important to the interpretation of the results. Soil data alone would show only the average lead concentration at various stages of each study. Soil data by study group would show apparent differences between groups, which by statistical inference might indicate the impact of intervention. When the soil data are linked to external dust or house dust, the impact of soil on dust, with or without intervention, becomes discernable. Through these data linkages, it is ultimately possible to construct a crude exposure scenario for the individual child. These scenarios begin with the simplest case, but can become complex in short order. For example, a young child may spent most of the time indoors, whereupon the exposure scenario becomes the lead that is available to the child through food, drinking water, air, and dust (see Figure 2-1). Each of these is influenced by one or more other sources of lead.

Most of the rest of this chapter consists of a discussion of specific data linkages that are arithmetic means of a specific compartment, such as soil, dust, hand dust, or blood lead, within study groups The reader is encouraged to become familiar with these graphical presentations of the data, because they form the basis of the statistical inferences of Chapter 4, and the conclusions of Chapter 5.

Data are also linked by a primary identifier or index. Some data are linked to the individual child, such as blood lead and hand lead. Some are specific for the living unit or family, and some are specific for the property. In Cincinnati, soil and exterior dust data are linked at the neighborhood level. It is important to be aware of this distinction because of the duplication effect that can occur when there are several siblings in a family and several families in a dwelling. This means that a single soil value could be heavily weighted if there were, for example, five children living on the same property.

#### 3.2.1.4 Data Transformations

***Processing Original Data Sets***

The data were received in their original form as personal computer compatible data sets constructed according to the data management procedures of the individual study. From the original data set, certain identifiers were removed to protect the privacy of the individuals and their families, and apparent discrepancies (missing or unused data) were resolved with the database managers for each study.

From this set of corrected original data, merged databases were formed in spreadsheet format, according to the primary index. There is one KID, FAM, and PROP file for each study, and a NBHD file for Cincinnati. Each of these intermediate files is large and cumbersome, but is amenable to extraction of information for specific statistical and data analysis purposes. The final condensed files used for this report consisted of selected fields from the intermediate files. Certain new fields were added for the convenience of data analysis. These were:

(1) a numeric value for the number of days since the start of the project (January 1, 1989),

(2) an arithmetic mean for soil and dust (discussed in Section 3.2.1.4), and

(3) the blood lead concentrations corrected for seasonal cycles and long-term trends (discussed in Section 3.3.5.1).

With these new fields, the condensed files were converted to SYSTAT format for the statistical analysis of the data as described in Chapter 4.

***Measures of Central Tendency for Soil and Dust***

For soil and dust, there is a need to reduce multiple measurements to a single representative data point for each property or living unit within a round. In order to determine the appropriate central tendency for this measurement, the participating groups discussed several alternatives at great length without reaching a consensus. Therefore, different measures of central tendency were reported in each of the three studies. The following is an extended discussion of each of these measures, followed by an argument for the use of the arithmetic mean as the best measure in these circumstances.

The procedures for selecting a representative soil sample were based on the statistical distribution of data in each study. The Boston study used the arithmetic mean, giving equal weight to all values. The Cincinnati study used the geometric mean, a method that is often used when the measured values are lognormally distributed because it gives lesser weight to extremes. The geometric mean is always lower than the arithmetic mean (except in the case of a perfectly normal distribution) and therefore may be an underestimate of the exposure to the child.

The distribution problem was approached differently in Baltimore, where the tri-mean was calculated as the weighted average of the first, second, and third quartiles:

$$X = \frac{Q_1 + 2Q_2 + Q_3}{4}, \qquad (3\text{-}1)$$

where $X$ = tri-mean, and
$Q_n$ = upper cutoff for the $n$th quartile

The tri-mean approach gives some consideration to the uneven distribution of values without unduly weighting the extremes. As with the geometric mean, the tri-mean is equivalent to the arithmetic mean if the distribution is perfectly normal. For distributions skewed to the left, the estimate is less than the arithmetic mean, and the estimate is greater than the arithmetic mean for right skewed distributions.

The ideal measurement of central tendency is one that perfectly represents exposure to the child. In this case a sample would be taken at each location where the child played and this sample would be weighted according to the time spent playing there and the frequency of hand-to-mouth activity during that time. Because this information is not available, a simplification assumption is that weight should be given to location rather that concentration because location, not lead concentration, is the basis of choice for the child's play environment.

All three approaches assume that the sampling pattern is random and that exposure to soil is spatially random. Neither condition is strictly true in all three studies. One-third to one-half of the soil samples were taken 1 m from the foundation of the home, where concentrations are known to be higher than elsewhere. Because of playtime interests, parental instructions, or other influences, the child tends to play in specific areas that may represent less than 25% of the total soil area.

It would seem reasonable that the ideal method for selecting a representative value should focus on the relationship between the soil and the child. In this respect, an exposure weighted mean of the soil samples would seem to be the most direct approach. This would be an arithmetic mean of soil values corrected for the degree of exposure to the child. For example, a sample taken from bare soil in an area observed to be a play area would be given a high weighting factor for exposure. Grass covered areas with limited accessibility would be weighted on the low end of exposure. Although cumbersome, this method is feasible because such information was collected at the time of sampling in each study. The drawback is that the method emphasizes the direct, outdoor playtime contact between the child and the soil, and does not consider other routes of dust exposure, such as soil ⇒ household dust.

An alternative solution is to consider that the child has equal exposure to the top 2 cm of the entire surface of the soil. In this case, the perfect sample would be to scrape up this upper 2 cm of soil, homogenize it and take a sample. Theoretically, this is equivalent to sampling in a random pattern and taking the arithmetic mean of these samples. In this project, random locations were taken along lines specifically selected to represent the expected high- and low-concentration areas of the plot of soil. In this sense, the arithmetic mean is the best measure of the central tendency of soil data, and is the statistic used in this report.

#### 3.2.1.5 Adjustments and Corrections to the Data

***Subjects Dropped from Study***

During the analysis of their data, the Boston group discovered that two children of the same family had apparently become exposed to lead-based paint while staying at a house outside their neighborhood during a time when it was being remodeled. Both siblings had blood lead concentrations that had tripled in less than 5 mo, between Rounds 2 and 3, from 10 to 35 and 17 to 43 $\mu$g/dL. The Boston group analyzed their data with and without these children, eventually excluding these data from the analyses used to confirm their hypothesis. This report accepts the conclusion that the data are outliers and dropped them from further analysis. There were no other individuals, families, or properties excluded as outliers from any of the three studies.

***Unit Conversion***

All data were converted to common units, usually metric. No further corrections were made for analytical blanks or similar analytical adjustments, other than as reported by each study.

***Missing Data***

The Baltimore data set for children's blood lead contained a large number of missing data. This was because of the complexity of scheduling clinic visits for large numbers of children over an extended period of time. Statistical procedures for imputing values for these data were used and the results of the reanalysis are given in Chapter 4. Although these imputed data would have changed the conclusions slightly, this procedure for imputing missing data was not used in reaching the primary conclusions of this report. Such procedures for imputing data are becoming more acceptable, and the reader should be aware of their possible impact.

*Seasonal Cycles*

In the review of the individual reports, the participants were encouraged to explore possible indicators of seasonal and long-term effects on children's blood lead. None were discovered in the individual study reports. However, from the vantage point of the three studies viewed side by side, it is apparent that there is a persistent seasonal pattern for blood lead concentrations that is consistent among all three studies and that can be normalized to reduce the impact of blood measurements taken over a span of weeks during each round. The normalizing parameters appear to be independent of obvious environmental factors.

Furthermore, there appears also to be a long-term downward trend in blood lead similar to that observed in other studies. The slope of this downward trend was not the same for all three studies. The discussion of the mathematical methods for making these two corrections to the blood lead concentrations is presented in Section 3.3.5.1.

## 3.3 PRESENTATION OF THE DATA

The purpose of this section is to visually present the data in a manner that illustrates apparent relationships between lead and the child's environment. For each set of figures there is a conclusion drawn in Section 3.4 and supporting statistical analyses in Chapter 4. This approach is intended to give the reader of Chapter 3 an opportunity to digest the data and preliminary hypotheses, then accept or reject these hypotheses based on the statistical analyses and inferences in Chapter 4.

The summarized data for all three studies used in this reanalysis appear in Tables 3-7, 3-8, and 3-9. For the most part, these data are the basis for the statistical analyses in Chapter 4.

TABLE 3-7. SUMMARY OF BOSTON STUDY DATA

| | ROUND 1 | ROUND 2 | ROUND 3 | ROUND 4 |
|---|---|---|---|---|
| Mean Soil Pb Conc. (μg/g) | | | | |
| BOS SPI | 2,605 | | 141 | 232 |
| BOS PI | 2,822 | 2,780 | 2,662 | 2,502 |
| BOS P | 2,780 | 2,780 | 2,729 | 2,679 |
| Mean Soil Pb Conc. >2,500 (μg/g) | | | | |
| BOS SPI | 3,167 | | 142 | 282 |
| BOS PI | 3,471 | 3,518 | 3,193 | 2,914 |
| BOS P | 3,518 | 3,518 | 3,440 | 3,362 |
| Mean Floor Dust Pb Conc. (μg/g) | | | | |
| BOS SPI | 6,761 | 2,445 | 3,239 | 1,311 |
| BOS PI | 4,202 | 1,763 | 1,476 | 1,337 |
| BOS P | 5,231 | 3,337 | 1,443 | 1,863 |
| Mean Floor Dust Load (mg/m²) | | | | |
| BOS SPI | 51 | 53 | 54 | 27 |
| BOS PI | 39 | 31 | 39 | 30 |
| BOS P | 47 | 47 | 47 | 30 |
| Mean Floor Dust Pb Load (μg/m²) | | | | |
| BOS SPI | 352 | 148 | 245 | 39 |
| BOS PI | 117 | 50 | 51 | 39 |
| BOS P | 291 | 185 | 79 | 67 |
| Mean Window Dust Pb Conc. (μg/g) | | | | |
| BOS SPI | 10,343 | 5,452 | 6,906 | 11,529 |
| BOS PI | 10,393 | 2,304 | 6,350 | 12,184 |
| BOS P | 13,030 | 11,414 | 9,799 | 13,171 |
| Mean Window Dust Load (mg/m²) | | | | |
| BOS SPI | 0.140 | 0.079 | 0.176 | 0.190 |
| BOS PI | 0.182 | 0.033 | 0.171 | 0.226 |
| BOS P | 0.176 | 0.165 | 0.155 | 0.231 |
| Mean Window Dust Pb Load (μg/m²) | | | | |
| BOS SPI | 3.11 | 1.22 | 2.09 | 2.87 |
| BOS PI | 6.30 | 0.22 | 2.22 | 4.29 |
| BOS P | 10.97 | 7.04 | 3.11 | 3.92 |
| Mean Hand Pb Load (μg/pair) | | | | |
| BOS SPI | 14.88 | | 14.59 | 18.08 |
| BOS PI | 13.97 | 14.88 | 14.44 | 18.10 |
| BOS P | 14.88 | 14.88 | 16.18 | 21.99 |
| Mean Blood Pb Conc. (μg/dL) | | | | |
| BOS SPI | 13.10 | | 10.19 | 10.65 |
| BOS PI | 12.37 | 12.02 | 8.85 | 11.49 |
| BOS P | 12.02 | 12.02 | 9.83 | 11.35 |
| GM Corrected Blood Pb Conc. (μg/dL) | | | | |
| BOS SPI | 11.52 | | 11.28 | 10.22 |
| BOS PI | 11.52 | 11.52 | 10.30 | 11.07 |
| BOS P | 11.52 | 11.52 | 11.52 | 11.52 |

TABLE 3-8. SUMMARY OF BALTIMORE STUDY DATA

| | Round 1 | Round 2 | Round 3 | Round 4 | Round 5 | Round 6 |
|---|---|---|---|---|---|---|
| Mean Soil Pb Conc. ($\mu$g/g) | | | | | | |
| BAL SP | 501 | N.D.[a] | N.D. | 36 | N.D. | N.D. |
| BAL P-1 | 552 | N.D. | N.D. | N.D. | N.D. | N.D. |
| BAL P-2 | 402 | N.D. | N.D. | N.D. | N.D. | N.D. |
| Mean Hand Pb Load ($\mu$g/pair) | | | | | | |
| BAL SP | 10.7 | 12.9 | 7.4 | 8.5 | 12.6 | 14.9 |
| BAL P-1 | 13.6 | 14.8 | 9.5 | 6.0 | 17.3 | 13.0 |
| BAL P-2 | 9.9 | 13.7 | 9.0 | 6.5 | 15.5 | 13.0 |
| Mean Blood Pb Conc. ($\mu$g/dL) | | | | | | |
| BAL SP | 12.0 | 11.0 | 10.5 | 9.5 | 11.1 | 10.6 |
| BAL P-1 | 12.7 | 11.8 | 10.1 | 8.9 | 9.3 | 9.7 |
| BAL P-2 | 12.5 | 11.3 | 12.0 | 10.1 | 10.8 | 10.6 |
| GM Corrected Blood Pb Conc. ($\mu$g/dL) | | | | | | |
| BAL SP | 10.0 | 10.8 | 10.7 | 10.3 | 9.8 | 9.0 |
| BAL P-1 | 10.5 | 11.4 | 9.5 | 9.5 | 8.6 | 8.0 |
| BAL P-2 | 10.1 | 11.2 | 11.4 | 11.4 | 10.1 | 9.3 |

[a]N.D. = Not determined.

### 3.3.1 Effectiveness and Persistency of Soil Abatement

Soil abatement took place between two rounds of soil measurements. Figure 3-10 visually illustrates the concept that preabatement soil concentrations remained constant until abatement, then decreased abruptly during abatement to the level measured at the next round. The reader should understand that these extrapolations may not represent the actual rate at which concentrations changed, but likewise, it would be inappropriate to assume gradual change suggested by the dashed line between the preabatement and first postabatement measurements and to attach some significance to the slope. This issue becomes important again in Sections 3.3.4 and 3.3.5 when the impact of intervention on hand and blood lead is discussed.

## TABLE 3-9. SUMMARY OF CINCINNATI STUDY DATA

| | Round 1 | Round 2 | Round 3 | Round 5 | Round 6 | Round 7 | Round 9 |
|---|---|---|---|---|---|---|---|
| Mean Soil Pb Conc. ($\mu$g/g) | | | | | | | |
| CIN SEI | 680 | 134 | 142 | 103 | 122 | 166 | 132 |
| CIN I-SE-1 | 237 | 247 | 240 | 262 | 125 | 182 | 138 |
| CIN I-SE-2 | N.D. | 1,012 | 555 | 724 | 233 | 251 | 442 |
| CIN NT | 339 | 346 | 330 | 256 | 331 | 267 | 266 |
| Mean Street Dust Pb Conc. ($\mu$g/g) | | | | | | | |
| CIN SEI | 3,937 | 3,398 | 2,118 | 2,559 | 3,231 | | |
| CIN I-SE-1 | 3,665 | 3,416 | 3,411 | 2,275 | 3,040 | | |
| CIN I-SE-2 | 3,644 | 1,990 | 1,920 | 1,680 | 2,905 | | |
| CIN NT | 1,583 | 1,156 | 891 | 968 | 1,086 | | |
| Mean Street Dust Load (mg/m$^2$) | | | | | | | |
| CIN SEI | 454 | 242 | 363 | 452 | 310 | | |
| CIN I-SE-1 | 649 | 561 | 326 | 420 | 126 | | |
| CIN I-SE-2 | 760 | 726 | 533 | 508 | 134 | | |
| CIN NT | 624 | 755 | 481 | 477 | 654 | | |
| Mean Street Dust Pb Load ($\mu$g/m$^2$) | | | | | | | |
| CIN SEI | 1,162 | 789 | 641 | 968 | 808 | | |
| CIN I-SE-1 | 2,364 | 1,618 | 1127 | 943 | 371 | | |
| CIN I-SE-2 | 2,440 | 973 | 739 | 648 | 302 | | |
| CIN NT | 1,005 | 957 | 498 | 587 | 442 | | |
| Mean Floor Dust Pb Conc. ($\mu$g/g) | | | | | | | |
| CIN SEI | 438 | 439 | 468 | 2,530 | 2,247 | | |
| CIN I-SE-1 | 417 | 512 | 478 | 2,924 | 2,040 | | |
| CIN I-SE-2 | 447 | 513 | 431 | 694 | 1,117 | | |
| CIN NT | 295 | 290 | 241 | 1,984 | 2,369 | | |
| Mean Floor Dust Load (g/m$^2$) | | | | | | | |
| CIN SEI | 1.67 | 0.77 | 0.28 | 0.82 | 0.51 | | |
| CIN I-SE-1 | 0.82 | 0.09 | 0.16 | 0.67 | 0.54 | | |
| CIN I-SE-2 | 0.82 | 0.21 | 0.20 | 3.28 | 0.33 | | |
| CIN NT | 0.44 | 0.47 | 0.20 | 0.64 | 0.29 | | |
| Mean Floor Dust Pb Load ($\mu$g/m$^2$) | | | | | | | |
| CIN SEI | 802 | 350 | 176 | 2,456 | 1,467 | | |
| CIN I-SE-1 | 415 | 55 | 76 | 1,356 | 1,059 | | |
| CIN I-SE-2 | 288 | 37 | 88 | 1,528 | 482 | | |
| CIN NT | 125 | 69 | 51 | 1,085 | 1,675 | | |

TABLE 3-9 (cont'd). SUMMARY OF CINCINNATI STUDY DATA

| | Round 1 | Round 2 | Round 3 | Round 5 | Round 6 | Round 7 | Round 9 |
|---|---|---|---|---|---|---|---|
| Mean Window Dust Pb Conc. ($\mu g/g$) | | | | | | | |
| CIN SEI | 1,807 | 1,493 | 1,210 | 5,080 | 4,033 | | |
| CIN I-SE-1 | 4,282 | 2,312 | 1,611 | 3,458 | 4,014 | | |
| CIN I-SE-2 | 2,622 | 1,900 | 1,532 | 5,940 | 4,390 | | |
| CIN NT | 1,975 | 1,443 | 992 | 2,982 | 2,412 | | |
| Mean Window Dust Load ($g/m^2$) | | | | | | | |
| CIN SEI | 10.3 | 1.86 | 0.52 | 17 | 6.56 | | |
| CIN I-SE-1 | 2.64 | 3.3 | 0.87 | 12.6 | 4.62 | | |
| CIN I-SE-2 | 5.57 | 2.71 | 0.71 | 11.0 | 6.0 | | |
| CIN NT | 13 | 13.8 | 0.73 | 14.5 | 5.3 | | |
| Mean Window Dust Pb Load ($\mu g/m^2$) | | | | | | | |
| CIN SEI | 33,700 | 135,000 | 1,413 | 60,990 | 20,500 | | |
| CIN I-SE-1 | 11,400 | 5,260 | 1,560 | 50,400 | 26,600 | | |
| CIN I-SE-2 | 47,400 | 4,600 | 1,220 | 198,000 | 34,500 | | |
| CIN NT | 22,700 | 18,500 | 726 | 112,000 | 24,500 | | |
| Mean Mat Dust Pb Conc. ($\mu g/g$) | | | | | | | |
| CIN SEI | 150 | 913 | 827 | 4,284 | 3,340 | | |
| CIN I-SE-1 | 215 | 1,950 | 769 | 2,271 | 2,423 | | |
| CIN I-SE-2 | 204 | 1,163 | 997 | 1,121 | 1,017 | | |
| CIN NT | 174 | 435 | 391 | 3,692 | 2,619 | | |
| Mean Mat Dust Load ($mg/m^2/day$) | | | | | | | |
| CIN SEI | | 6.5 | 7.7 | 4.4 | 28.2 | | |
| CIN I-SE-1 | | 18.7 | 4.7 | 4.9 | 16.6 | | |
| CIN I-SE-2 | | 8.5 | 7.9 | 6.2 | 3.6 | | |
| CIN NT | | 1.8 | 2.0 | 2.7 | 12.2 | | |
| Mean Mat Dust Pb Load ($\mu g/m^2/day$) | | | | | | | |
| CIN SEI | | 6.54 | 7.62 | 2.38 | 9.80 | | |
| CIN I-SE-1 | | 7.65 | 5.14 | 3.20 | 8.02 | | |
| CIN I-SE-2 | | 5.83 | 8.04 | 6.24 | 2.06 | | |
| CIN NT | | 3.30 | 4.67 | 0.99 | 5.29 | | |
| Mean Entry Dust Pb Conc. ($\mu g/g$) | | | | | | | |
| CIN SEI | 456 | 789 | 569 | 1,057 | 1,767 | 676 | 2,148 |
| CIN I-SE-1 | 452 | 812 | 456 | 819 | 922 | 1,507 | 1,789 |
| CIN I-SE-2 | 665 | 681 | 833 | 1,127 | 1,224 | 862 | 675 |
| CIN NT | 348 | 475 | 369 | 1,804 | 1,534 | 1,419 | 2,061 |

TABLE 3-9 (cont'd). SUMMARY OF CINCINNATI STUDY DATA

| | Round 1 | Round 2 | Round 3 | Round 5 | Round 6 | Round 7 | Round 9 |
|---|---|---|---|---|---|---|---|
| Mean Entry Dust Load ($g/m^2$) | | | | | | | |
| CIN SEI | 36.4 | 6.1 | 4.8 | 6.6 | 2.6 | 0.34 | 1.8 |
| CIN I-SE-1 | 1.0 | 0.11 | 0.50 | 10.9 | 1.07 | 0.64 | 2.24 |
| CIN I-SE-2 | 1.24 | 0.15 | 0.66 | 6.12 | 0.86 | 0.89 | 1.31 |
| CIN NT | 12.0 | 2.45 | 0.32 | 22.5 | 1.46 | 2.26 | 17.8 |
| Mean Entry Dust Pb Load ($\mu g/m^2$) | | | | | | | |
| CIN SEI | 16,800 | 5,670 | 2,856 | 9,517 | 3,634 | 238 | 3,042 |
| CIN I-SE-1 | 540 | 82 | 261 | 8,280 | 967 | 657 | 2,029 |
| CIN I-SE-2 | 828 | 144 | 496 | 9,678 | 1,186 | 589 | 1,695 |
| CIN NT | 6,715 | 513 | 113 | 23,300 | 4,800 | 1,270 | 12,900 |
| Mean Hand Pb Load ($\mu g$/pair) | | | | | | | |
| CIN SEI | 8.6 | 7.1 | 5.9 | 17.7 | 16.0 | 8.9 | 18.2 |
| CIN I-SE-1 | 12.3 | 8.3 | 6.5 | 9.0 | 10.4 | 7.5 | 16.3 |
| CIN I-SE-2 | 14.1 | 11.2 | 7.1 | 16.6 | 11.3 | 14.1 | 39.6 |
| CIN NT | 5.3 | 4.4 | 2.7 | 5.9 | 11.3 | 4.7 | 14.2 |
| Mean Blood Pb Conc. ($\mu g/dL$) | | | | | | | |
| CIN SEI | 10.4 | | 8.3 | 10.2 | | 9.3 | 9.9 |
| CIN I-SE-1 | 10.3 | | 8.8 | 8.1 | | 7.1 | 9.5 |
| CIN I-SE-2 | 13.7 | | 11.8 | 10.7 | | 9.8 | 12.1 |
| CIN NT | 9.2 | | 6.6 | 7.6 | | 7.2 | 7.9 |
| GM Corrected Blood Pb Conc. ($\mu g/dL$) | | | | | | | |
| CIN SEI | 8.3 | | 8.9 | 9.6 | | 11.8 | 12.0 |
| CIN I-SE-1 | 8.5 | | 9.3 | 8.5 | | 10.3 | 11.3 |
| CIN I-SE-2 | 9.8 | | 11.9 | 10.0 | | 12.1 | 13.6 |
| CIN NT | 7.3 | | 7.4 | 7.6 | | 9.9 | 10.7 |

In order to form an effective, permanent barrier between the source of lead and the human environment, soil abatement must reduce the concentration of lead in the soil in a manner that is persistent for a period of years. In each of the three studies, measurements were made prior to abatement and immediately after abatement (within 3 mo). Followup measurements were made periodically until the end of the study in Cincinnati and Boston. The results of these soil analyses, corrected for small errors, are graphically illustrated in Figures 3-11, 3-12, and 3-13. These data show, for all three studies, a substantial reduction

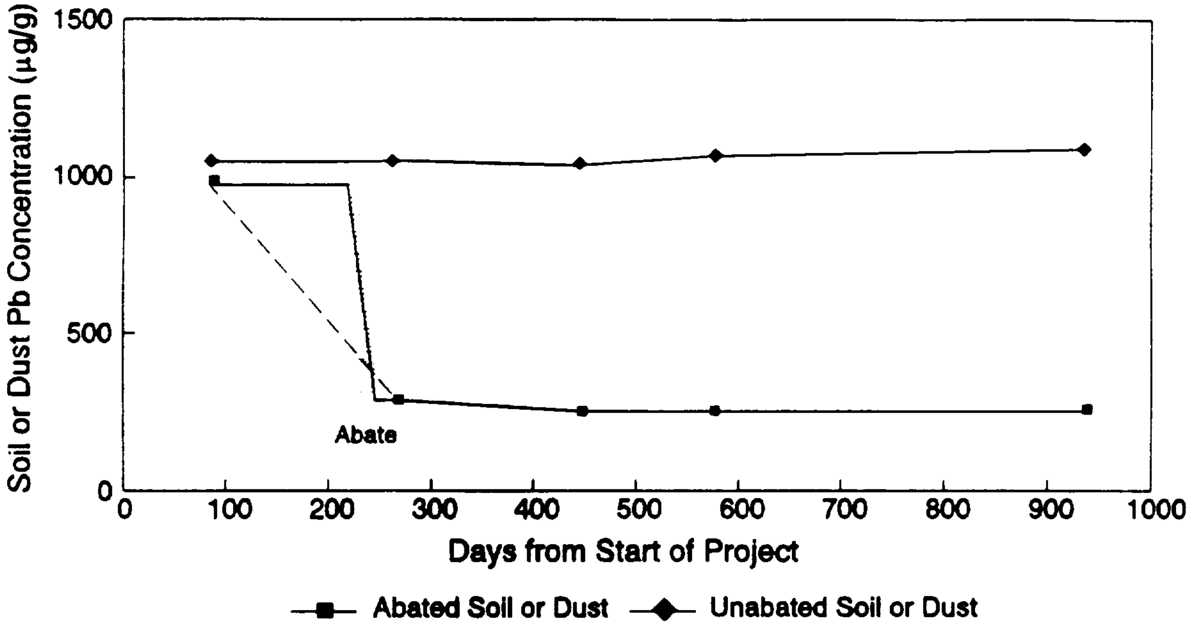


Figure 3-10. Hypothetical representation of intervention impact (solid lines, shaded areas) on soil and dust concentrations. When intervention occurs between rounds of measurements, the dashed line misrepresents the transition from the first to the second measurement in the abated group.

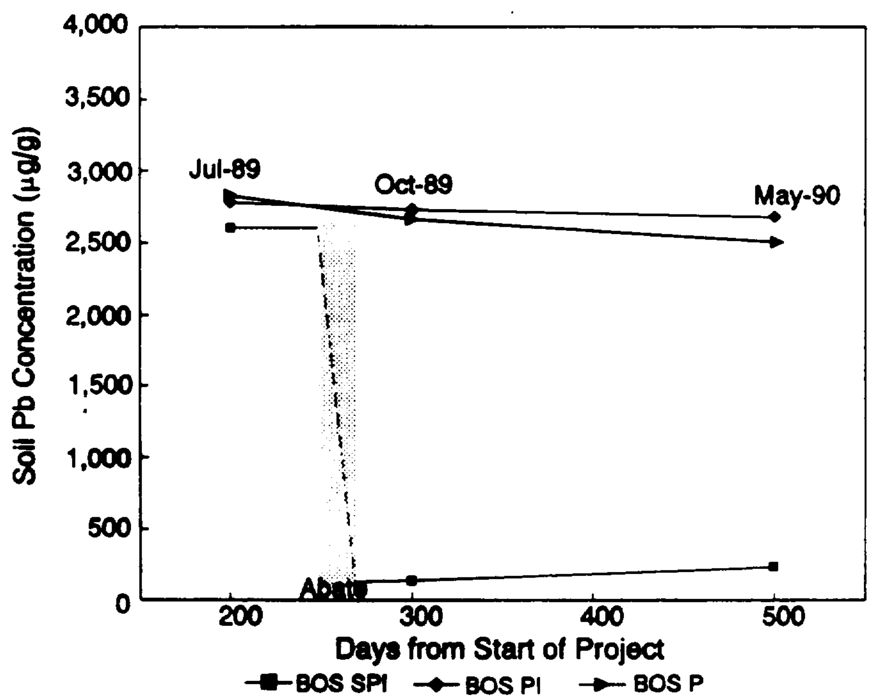


Figure 3-11. The arithmetic means of Boston soil lead concentrations by study group show the effectiveness and persistency of soil abatement. The date shown is the arithmetic mean of the individual site sampling dates. Figure 3-14 is a similar plot of soil lead concentrations above 2,500 µg/g.

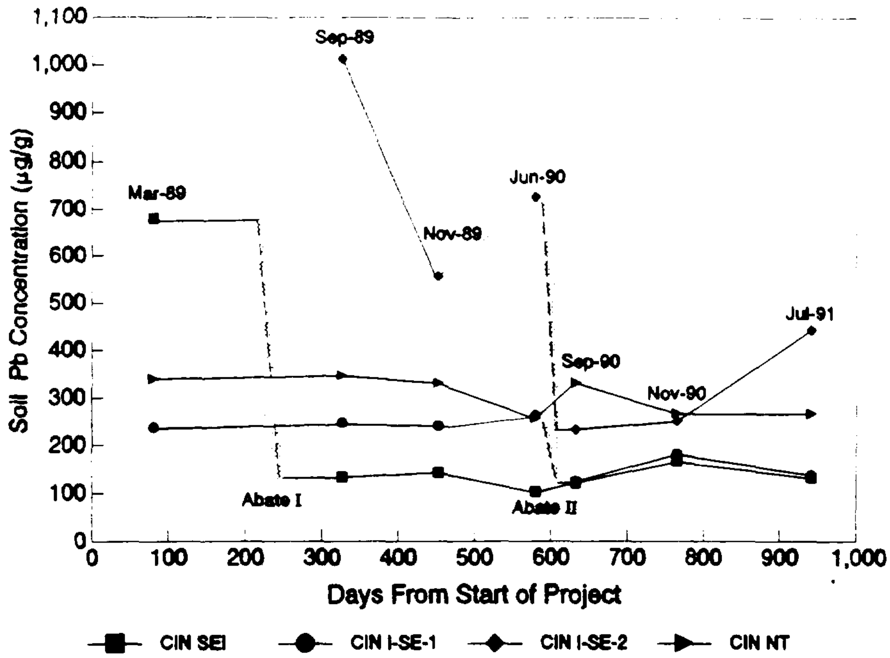


**Figure 3-12. Cincinnati soil lead concentrations. The arithmetic means by study group of the soil lead concentrations that show the effectiveness and persistency of soil abatement.**

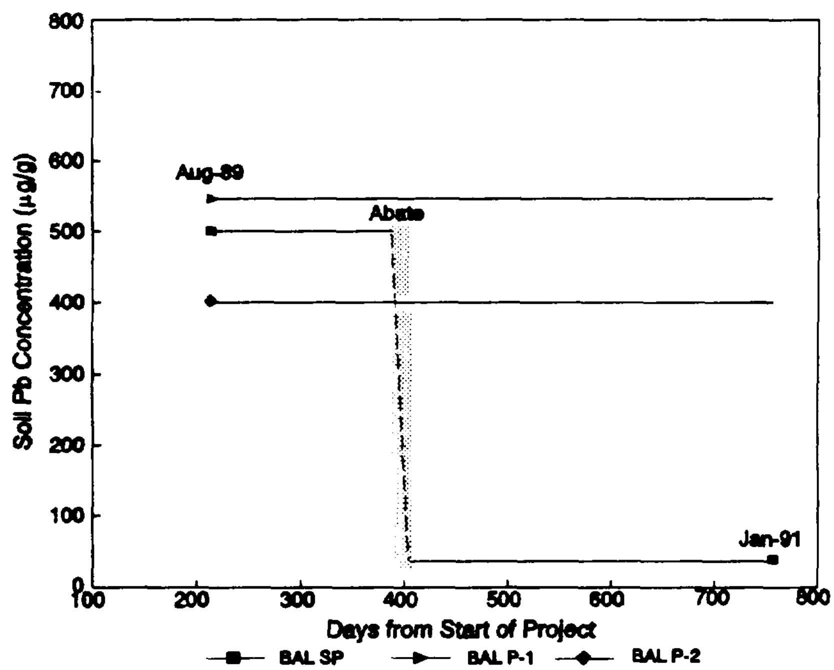


**Figure 3-13. Reconstruction of the expected effectiveness of soil abatement in the Baltimore study. Measurements were made for both study groups before abatement, but only for the BAL SP group after abatement. The lines for BAL P-1 and BAL-P-2 are shown only for comparison purposes.**

in the amount of lead in soil measured immediately after abatement, and that in Boston and Cincinnati, where followup soil measurements were taken, this reduction persisted for the duration of the study. For this calculation, the arithmetic mean was used in order to give equal weight to all soil samples taken. It should be noted that in Baltimore, the postabatement measurements were made only in the locations where soil had been excavated and removed.

Each study was able to achieve the targeted concentration for abated soil. The mean soil concentrations following abatement are not substantially higher than the specifications for clean soil. The amount of soil lead reduction actually achieved directly because influences the expected changes in dust lead and blood lead. In Section 3.3.5, an attempt will be made to evaluate the treatment/response relationship for each step of the pathway of lead in the human environment.

To determine the effectiveness and persistency of soil abatement, the arithmetic mean for each parcel of land was taken for each round where soil measurements were made. The parcel means for the Boston and Cincinnati studies show that abated soil concentrations (BOS SPI and CIN SEI) dropped significantly after abatement (Figures 3-11, 3-12, and 3-14), whereas unabated soil (BOS PI, BOS P, and CIN NT) appear to decrease only slightly, if at all. This reduction in the abated soil was persistent through the end of the study. The Cincinnati groups CIN SEI-1 and CIN SEI-2, which received soil and exterior dust abatement during the second year, showed a decrease in the range expected, but there were no followup measurements to demonstrate persistency. Because there were no measurements, in the Baltimore study, of abated soil beyond the first measurement immediately after abatement, and there were no measurements of unabated soil after the beginning of the study, the data show only that abatement was effective, with no measurement of persistency.

Because it is known that blood lead concentrations follow a seasonal cycle and a downward trend, every effort was made in this document to note and discuss possible trends or cycles in the environmental sources of lead that might explain this blood lead pattern.

There appears to be some indication of a general downward trend of lead concentrations in the unabated soil. Although not statistically significant for any individual group, the fact that all groups where the soil remained unabated show this phenomenon lends some credence

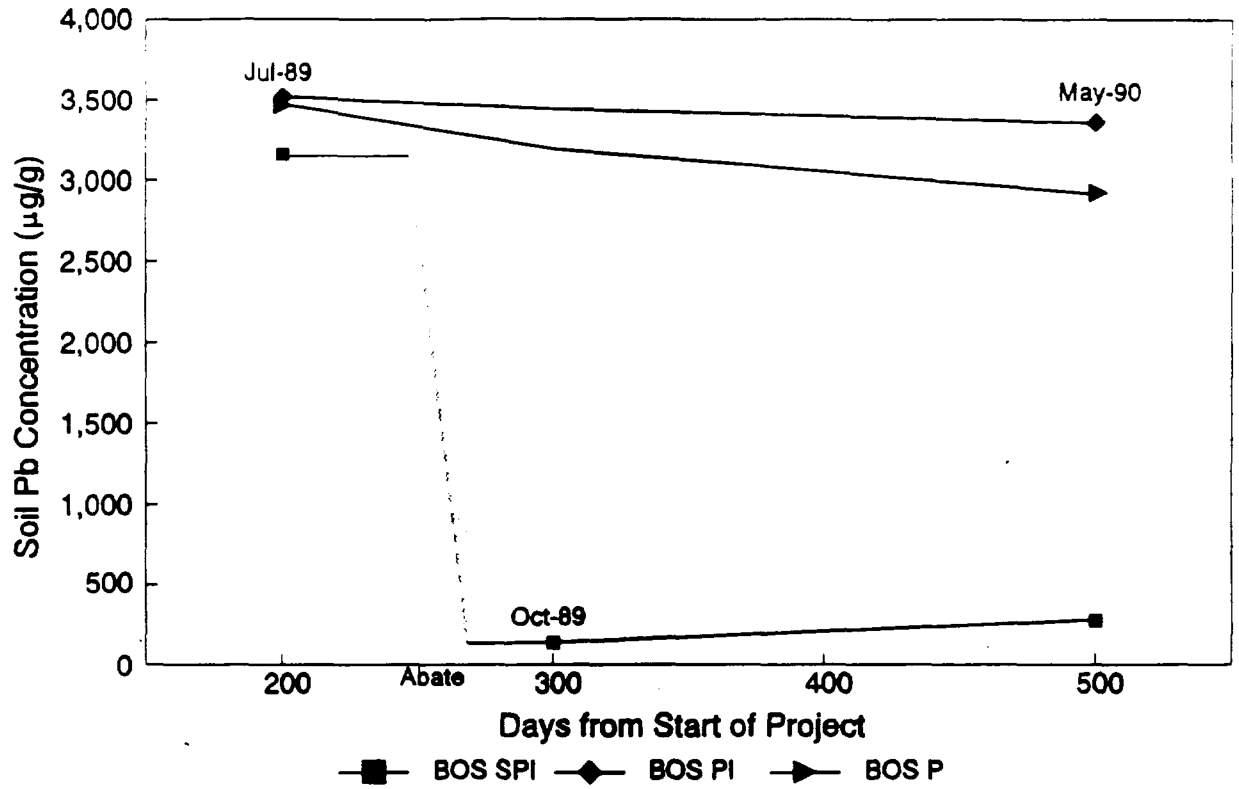


**Figure 3-14. Boston soil lead concentrations above 2,500 $\mu$g/g. Similar to Figure 3-11, these data show the effectiveness and persistency in the Boston study for soils with lead concentrations greater than 2,500 $\mu$g/g.**

to this observation. Analysis of quality assurance/quality control audit samples shows this trend cannot be attributed to analytical drift (see Section 3.1). There are several obstacles to demonstrating a temporal trend or cycle in soils. Soil lead concentrations vary widely over a relatively small distances, even less than 1 m. Because it was not feasible to return to the exact spot for sequential soil sample, it is not reasonable to expect two sequential samples to have the same value. It is reasonable to expect, however, that the differences with time to be random in the absence of a seasonal cycle or trend.

On the issue of recontamination following abatement, it is curious that such an increase did not occur in the comparable sites where no abatement occurred. This suggests that soil lead might reach some equilibrium with other components of the environment. Although this is not an unreasonable conclusion, there is no complete explanation for the mechanisms that regulate this equilibrium.

### 3.3.2 Effectiveness and Persistency of Exterior Dust Abatement

Dust is measured in both concentration and surface loading. Concentration is measured in micrograms of lead per gram of dust, whereas loading is measured in milligrams of lead per square meter. When dust abatement is performed, the amount of dust changes, but the concentration of lead in the dust does not. Therefore, there should be no change in dust lead concentration unless the source of the dust changes. Where soil abatement has been performed in connection with dust abatement, the dust lead concentration should also decrease abruptly. If there is a mixture of dust sources and only one has been abated, the lead concentration would change less abruptly, according to the contribution from each source.

Measurement of dust loading on smooth interior surfaces has in the past provided reliable information of the contribution of dust from multiple sources. The attempt to use this procedure for exterior dust in Cincinnati presented some problems. The surfaces such as asphalt and concrete were not as smooth as hardwood floors, and the sources were not as easily identified. These factors should be taken into consideration when interpreting the Cincinnati exterior dust data (Figures 3-15, 3-16, and 3-17).

In the Cincinnati study, the exterior dust load data (Figure 3-15) suggest a trend or cycle with a period of about 1 year for the unabated group (CIN NT). When this pattern is considered in the abated areas, it was not clear whether abatement of exterior dust was effective or whether the abated areas were recontaminated before the postabatement measurements were taken (1 week after abatement).

Exterior dust was measured and abated only in the Cincinnati study. In the CIN SEI group, exterior dust concentrations decreased after abatement, then increased steadily through the end of the study. In the CIN I-SE-1 group, exterior dust concentrations dropped similar to CIN SEI without abatement in 1989, then increased through the end of the study even following abatement in 1990. This indicates that even though the relative contribution of lead from other sources changed over time, exterior dust abatement did not seem to impact the contribution from these sources.

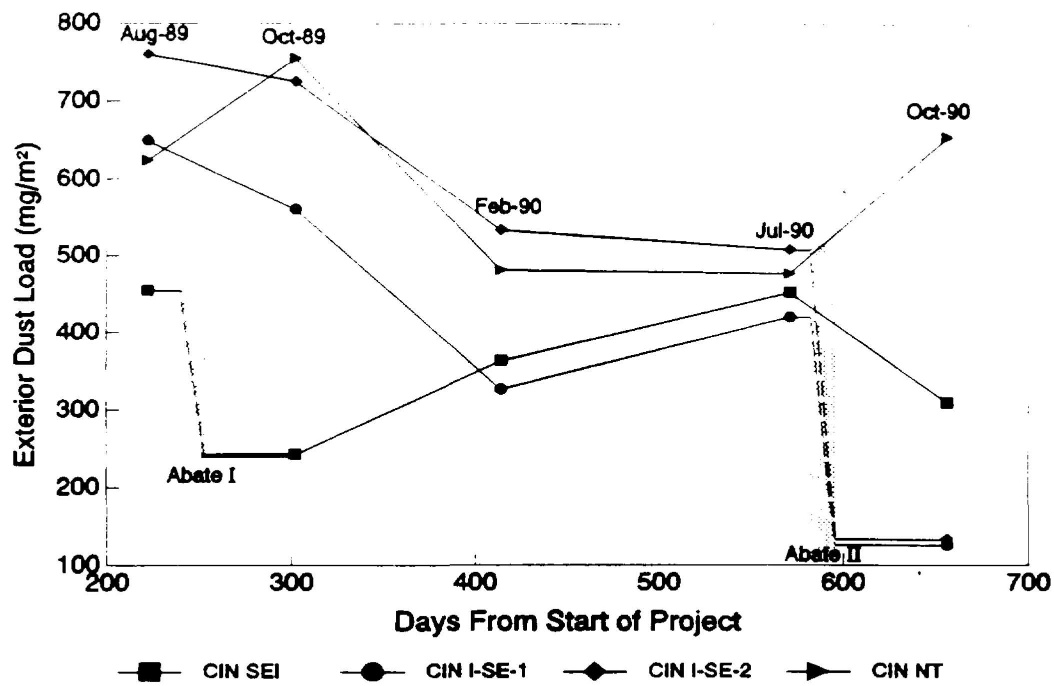


Figure 3-15. Cincinnati exterior dust load measurements. The data indicate that exterior dust abatement was effective but not persistent for more than 150 to 200 days.

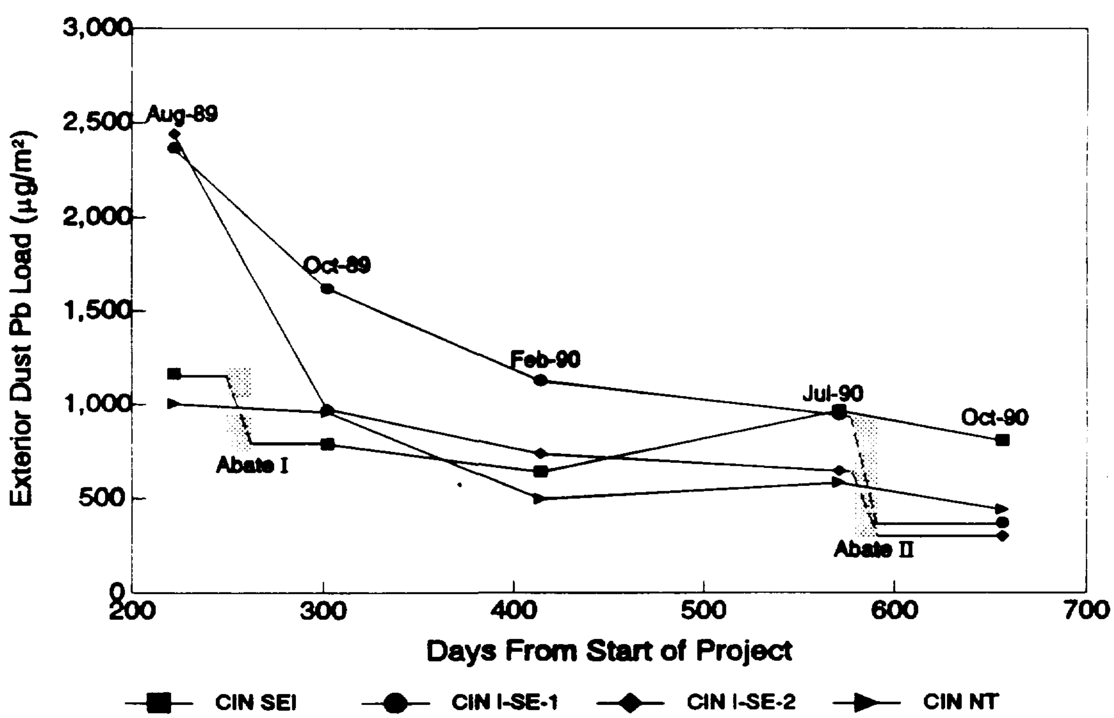


Figure 3-16. Cincinnati exterior dust lead load measurements. The data indicate small changes in the lead load that may have been persistent for several months, as indicated by the recovery time for CIN SEI of about 1 year.

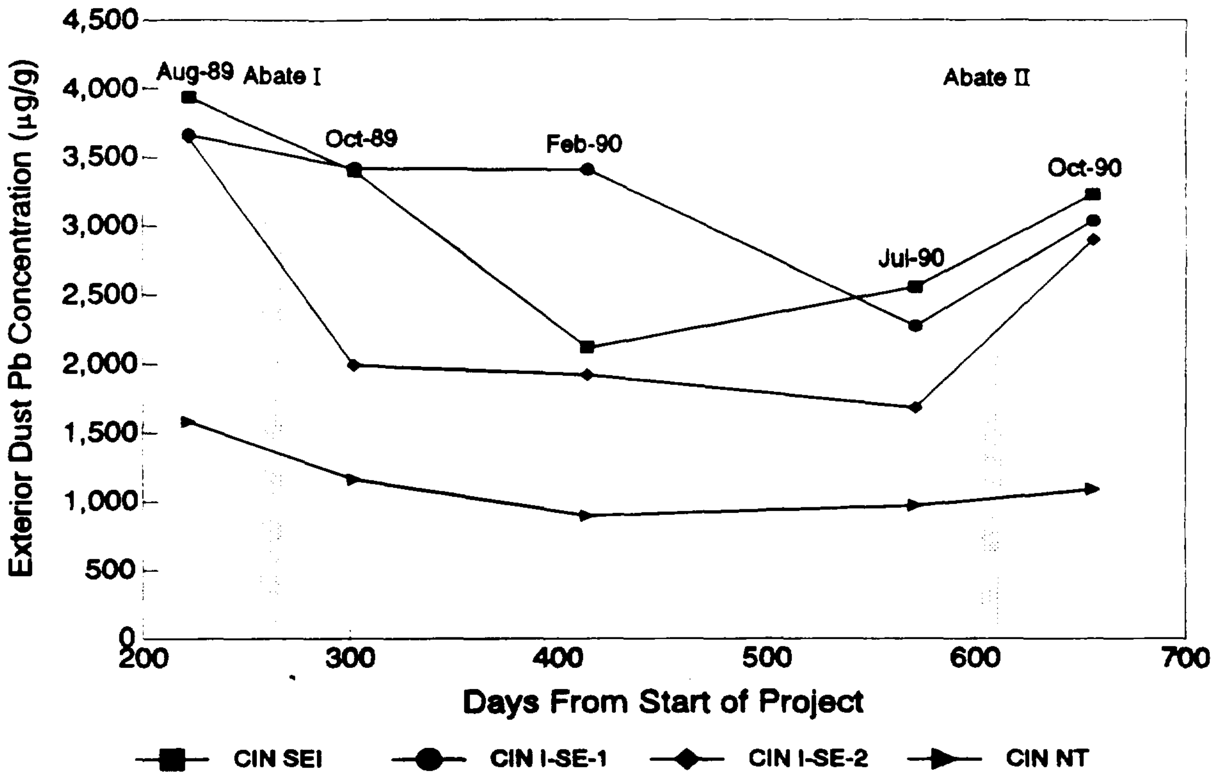


**Figure 3-17. Cincinnati exterior dust lead concentrations. Abatement is not expected to change the lead concentration unless the sources of lead are altered.**

Abatement of the exterior dust would not impact the lead concentration unless the sole source of the lead in exterior dust is soil and the soil parcels were abated prior to external dust abatement. Because the data for exterior dust do not reflect a response to soil abatement, yet do show a pattern of changing lead concentrations, it is reasonable to hypothesize another source for lead in exterior dust. For the purposes of this discussion, it is not necessary to identify this source, but it should be noted that the changes observed in exterior dust should also be reflected in the interior household dust. This question will be addressed in detail below.

As for Boston and Baltimore, the question arises that there may also be external sources of lead other than soil that contribute to household dust and to the exposure of children during outside activities. Because there were no measurements of exterior dust in these studies, little evidence is available to accept or reject this hypothesis. However, in the context of exposure pathways, the parcels of soil in Boston and Baltimore were on the individual properties, whereas in Cincinnati, they were in areas separated spatially from the living units, such as parks and vacant lots.

### 3.3.3 Effectiveness and Persistency of Interior Dust Abatement

The data for the Boston study interior dust are shown in Figures 3-18 through 3-23. In both BOS SPI and BOS PI, there was a general decrease in the floor dust lead loading following interior dust abatement, as shown in Figure 3-20, and further decreases were observed at 7 to 12 mo after abatement. In the window wells, however, the lead loading decreased immediately after dust abatement (Figure 3-23), persisted for a few months, then returned to original levels about 12 mo after abatement. The high concentrations of lead in the window well dust (5,000 to 22,000 $\mu$g/g) may indicate lead-based paint was present (Figure 3-22).

The Cincinnati study (Figures 3-24 through 3-35) found an immediate reduction in floor dust lead loading that persisted for at least 5 mo, followed by an increase at 12 mo to 70% of the preabatement level in CIN SEI, where soil abatement had taken place, and to nearly twice the preabatement level in CIN I-SE-1 and CIN I-SE-2, where soil had not yet been abated. Similar patterns were observed in the window wells (Figures 3-27 through 3-29) and entry ways (Figures 3-30 through 3-35). The window well concentrations were lower in Cincinnati (1,000 to 2,300 $\mu$g/g) than in Boston, suggesting a minimum influence of lead-based paint.

### 3.3.4 Hand Dust Results

Because hand-to-mouth activity is one route by which lead may be ingested, the amount of lead on the child's hand is an indicator of exposure. Only lead loading information is available because it was necessary to take the sample with wet wipes. The units of measurement are micrograms per pair of hands rather than micrograms per square meter. Nevertheless, these data are important intermediates between soil/dust and blood lead.

In Boston, there was a general increase in hand lead throughout the study (Figure 3-36). Although there is no explanation for this increase, there appears to be less of

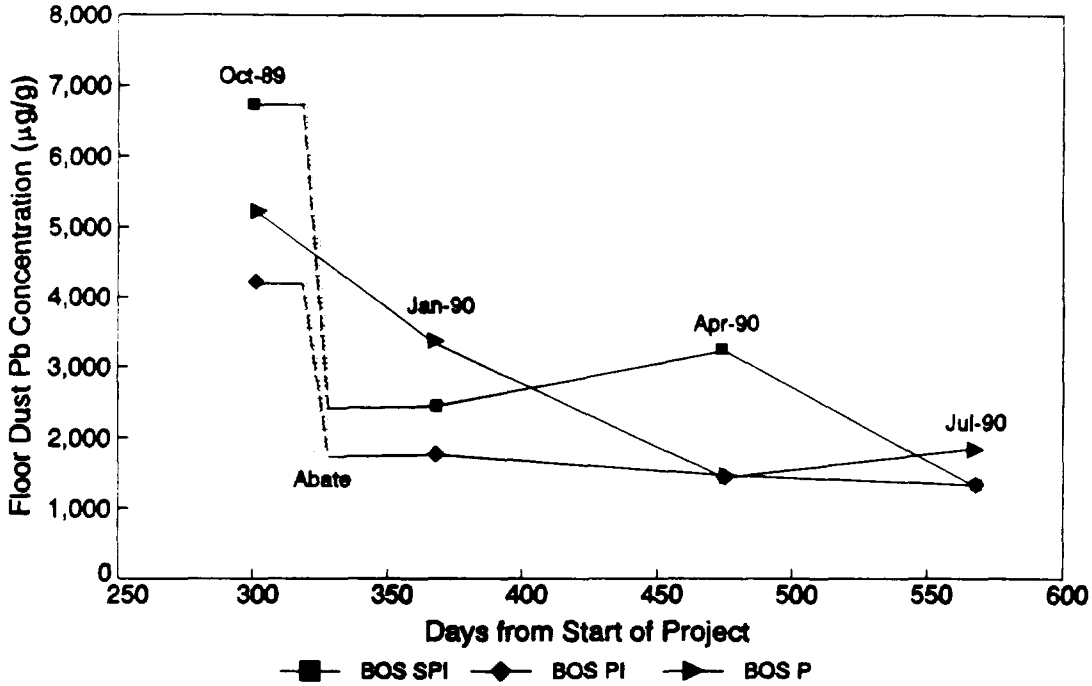


**Figure 3-18. Boston floor dust lead concentration. The data appear to support the observation that two sources of lead are present in household dust: soil and paint. Dust abatement alone is not expected to change the dust lead concentration.**

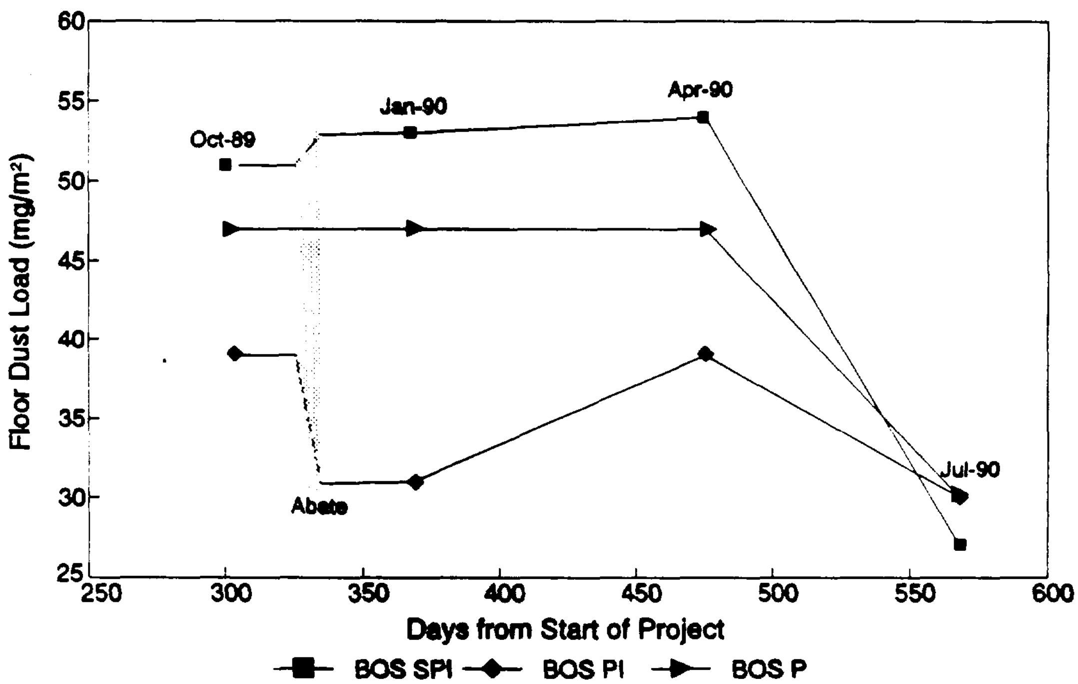


**Figure 3-19. Boston floor dust load. The dust load was not reduced in the BOS SPI group, indicating an immediate recovery. A longer recovery period is indicated for the BOS PI group.**

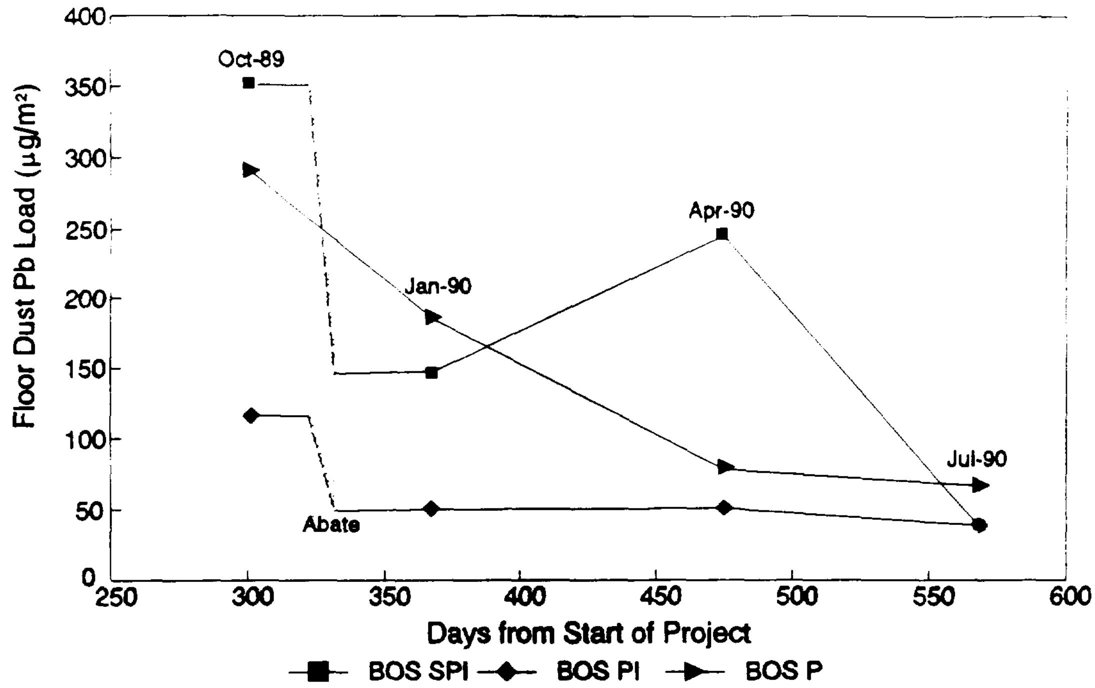


**Figure 3-20. Boston floor dust lead load. Even though the dust load in Figure 3-19 indicates a quick recovery, the lead load did not recover immediately, indicating that the source of the lead was cut off, at least temporarily.**

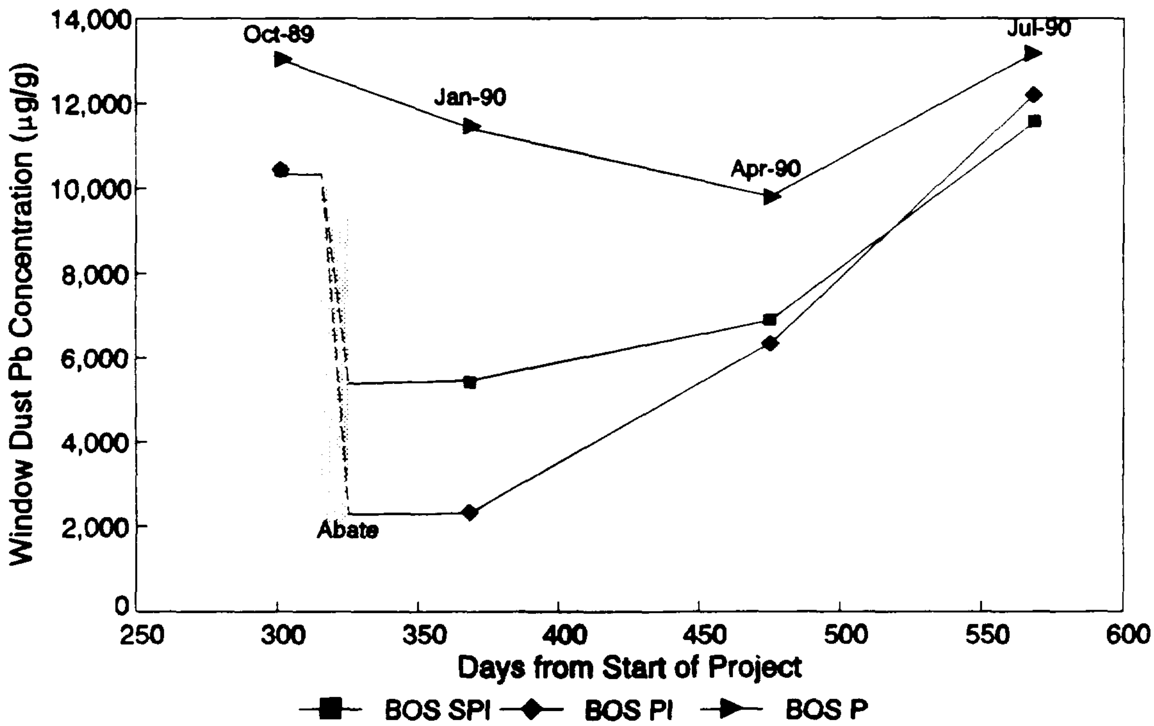


**Figure 3-21. Boston window dust lead concentrations. Paint stabilization and soil abatement appear to have been effective and persistent for several hundred days, similar to floor dust. The recovery observed between April and July 1990 was not observed for the floor dust load data.**

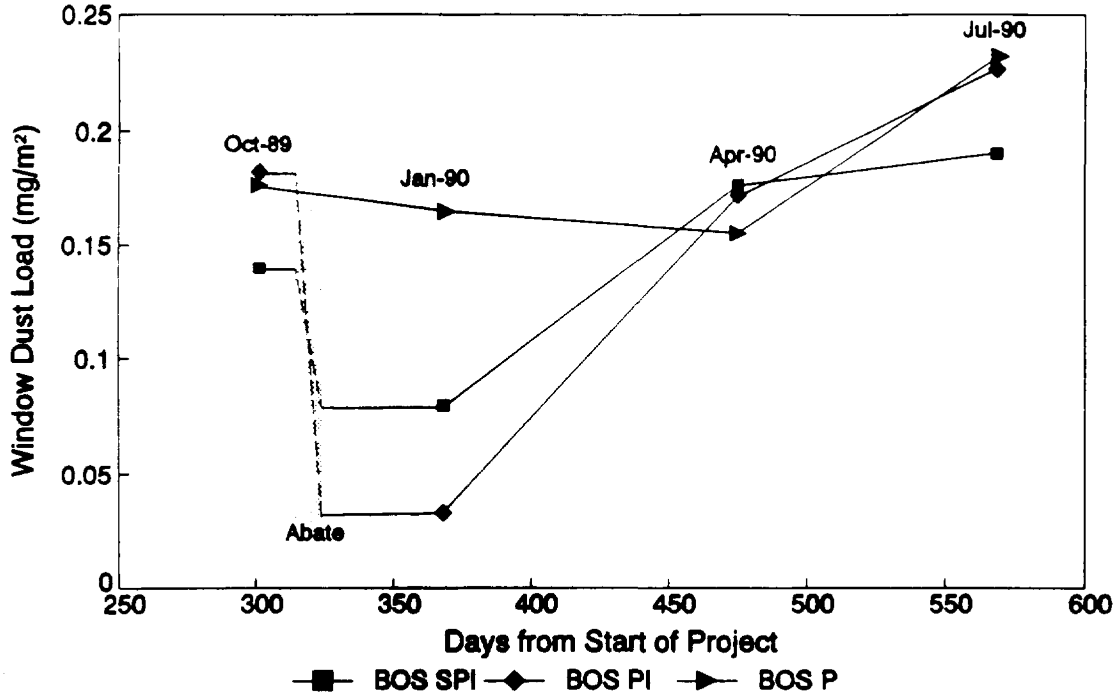


**Figure 3-22. Boston window dust load. These data show the effectiveness of window dust abatement, which appears to recover after about 150 days, similar to floor dust loads observed in Figure 3-19.**

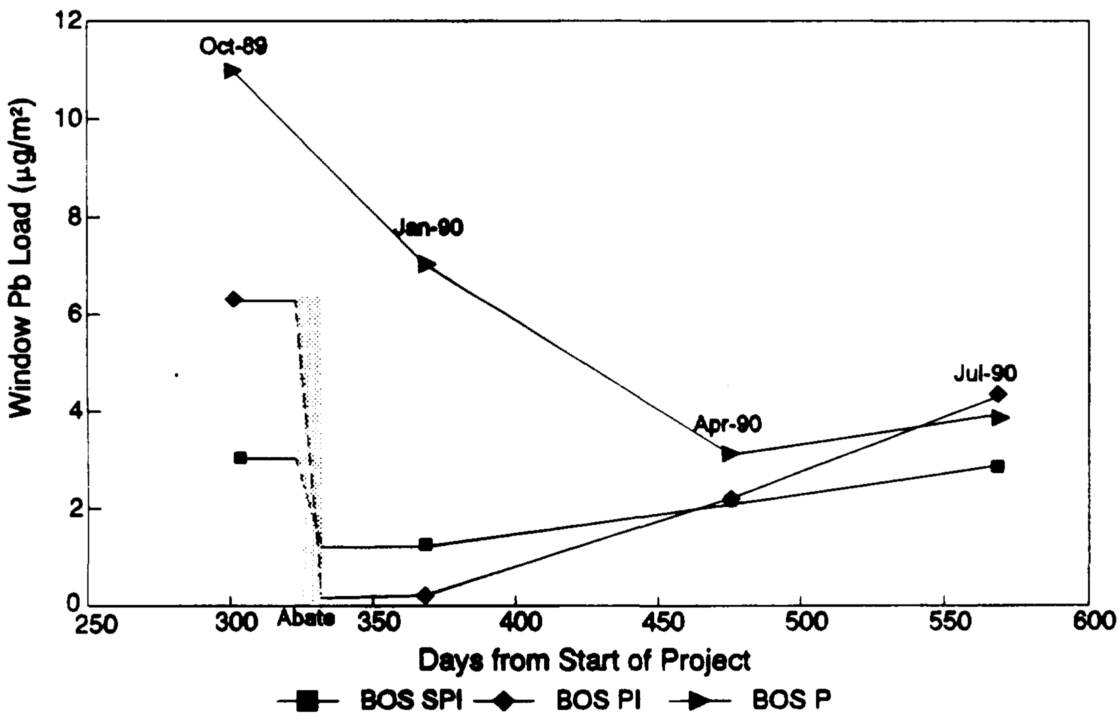


**Figure 3-23. Boston window dust lead load. As with floor dust lead loads, the window data indicate that both paint and soil sources of lead were interrupted, at least temporarily. The data appear to be consistent with Figure 3-20.**

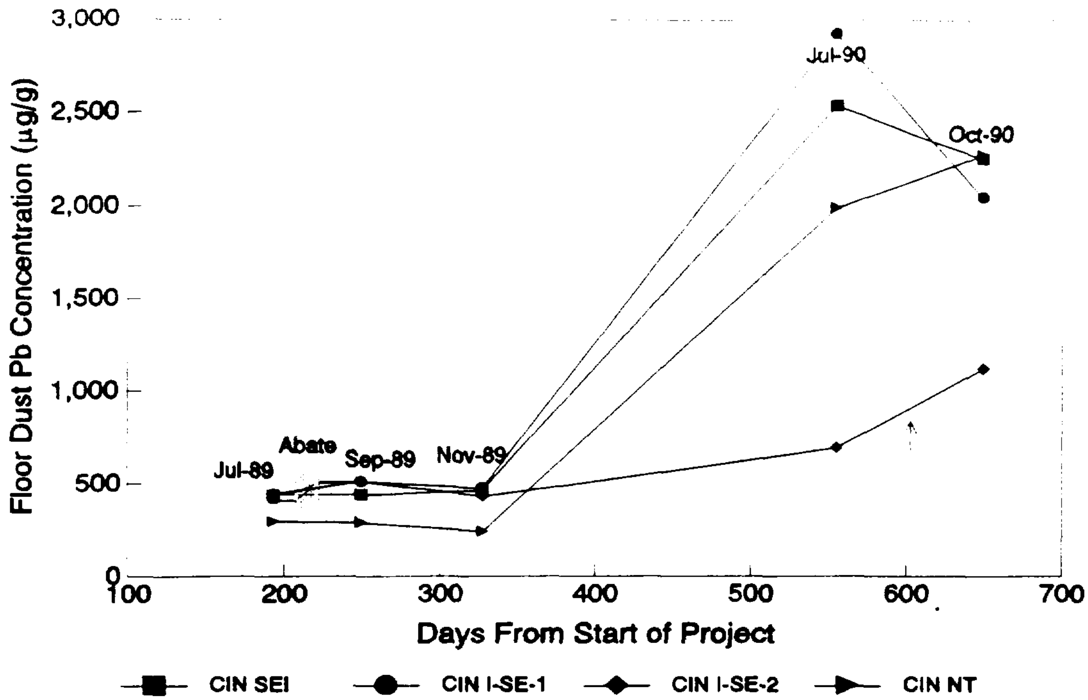


**Figure 3-24. Cincinnati floor dust lead concentrations. The small changes in lead concentrations suggest that the sources of lead did not change as a result of the abatement activities. The abrupt increase after November 1989 indicates one or more sources changed markedly during that time.**

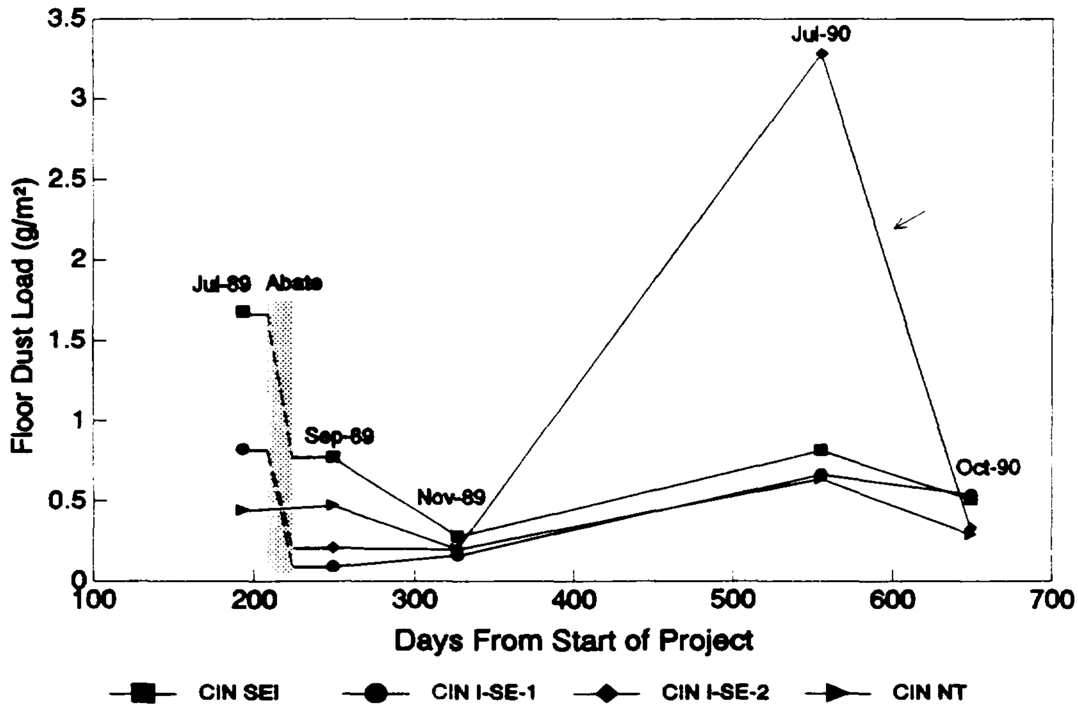


**Figure 3-25. Cincinnati floor dust load. These data confirm the effectiveness of the household dust abatement and show that this reduction was persistent for more than 100 days.**

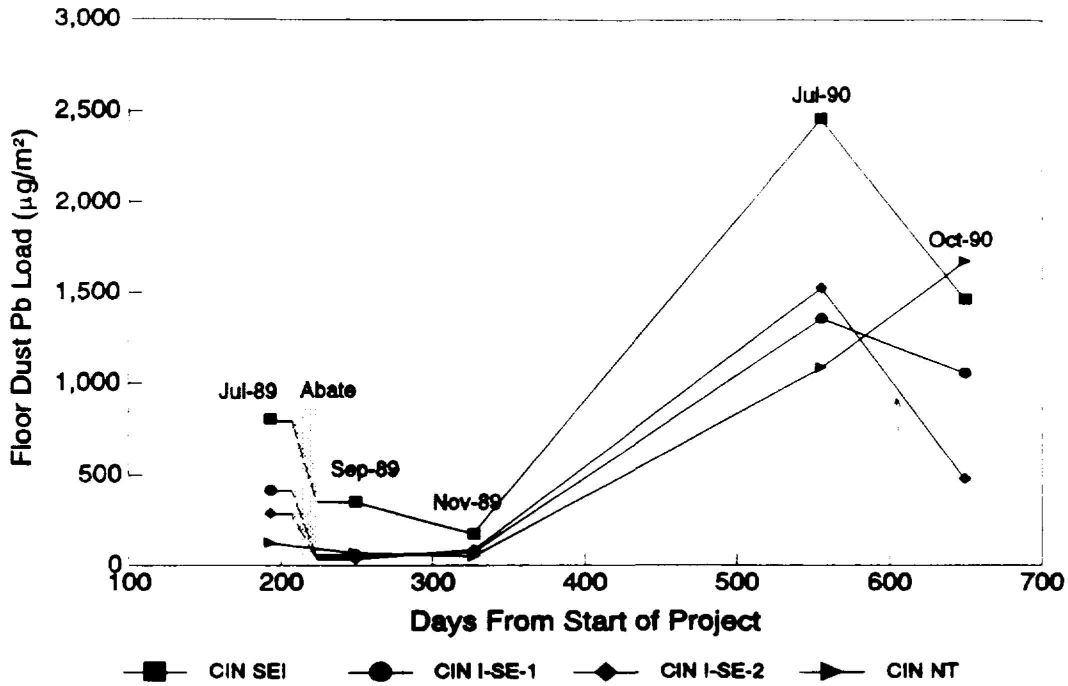


**Figure 3-26. Cincinnati floor dust lead load. The data suggest that the sources of lead were interrupted by the abatement activities, but that at least one source recovered after November 1989.**

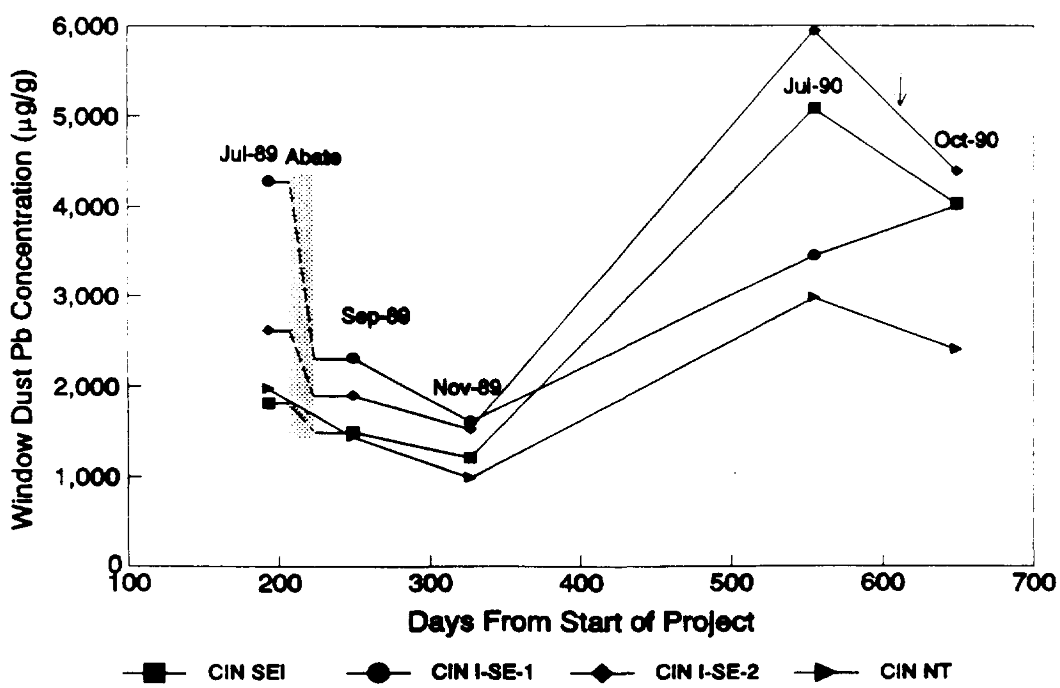


**Figure 3-27. Cincinnati window dust lead concentration. The response to abatement appears to be consistent with the observations of the floor dust in Figure 3-24.**

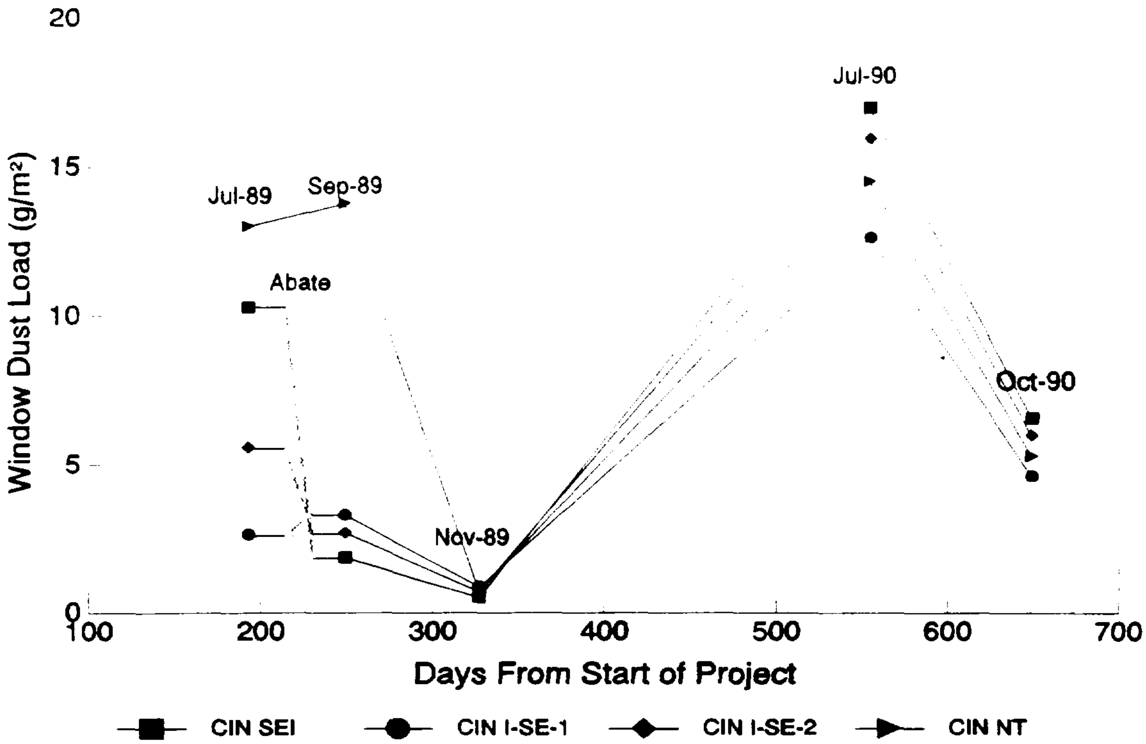


**Figure 3-28.** **Cincinnati window dust load. The impact of abatement and the change in the CIN NT group are consistent between floor dust load (Figure 3-25) and window dust load.**

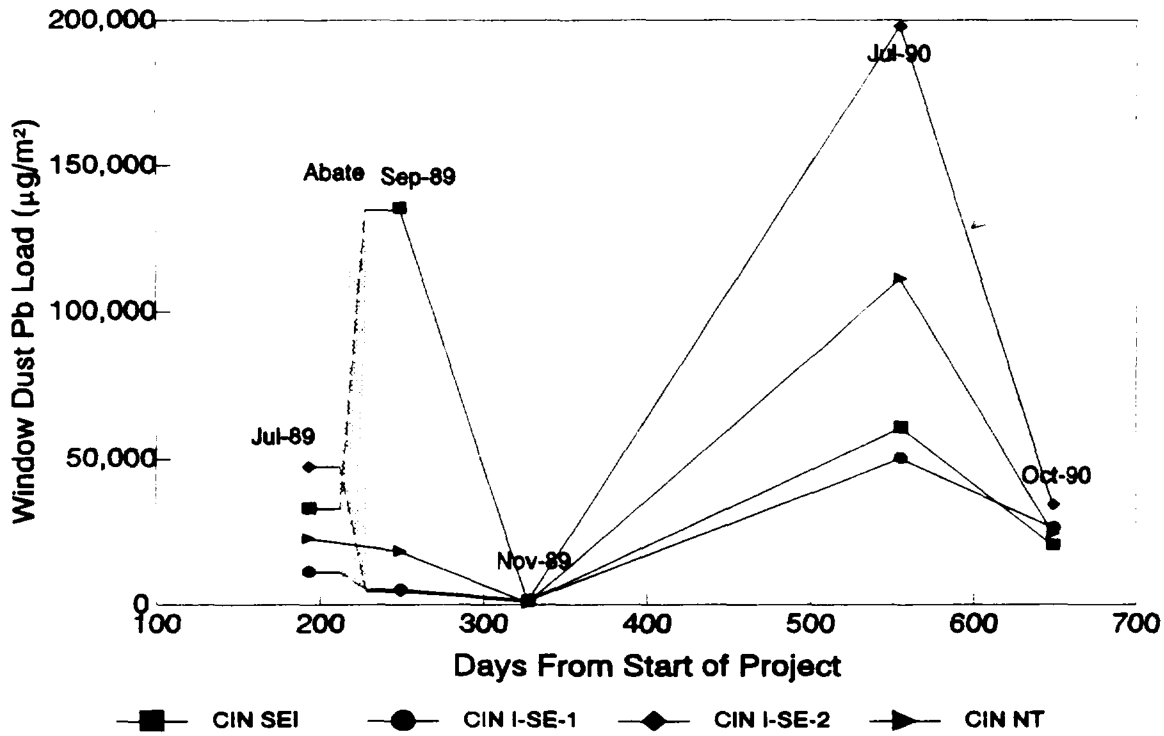


**Figure 3-29.** **Cincinnati window dust lead load. These data indicate one of the few instances where exposure to lead, as indicated by the household dust lead load, increased during abatement for one of the study groups (CIN SEI).**

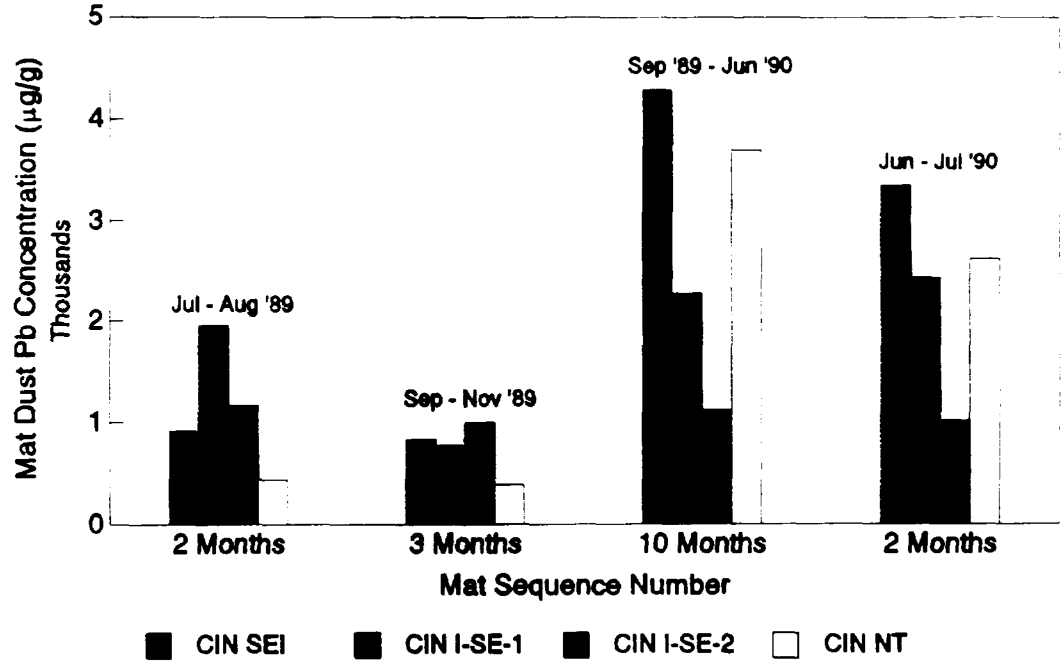


Figure 3-30. **Cincinnati mat dust lead concentration. Clean mats were placed at the entry to the housing unit. It was expected that the lead concentrations would reach equilibrium with the dust entering the home and remain constant for the duration of the study.**

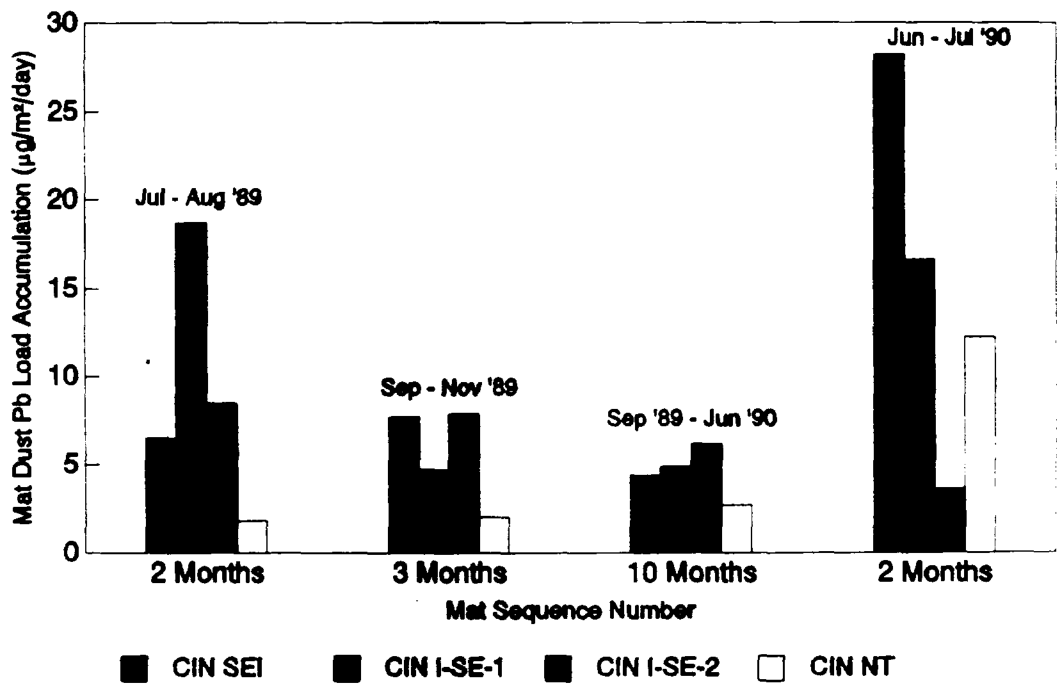


Figure 3-31. **Cincinnati mat dust load. In a general sense, the dust load should increase as long as the mats are in place, which appears to be the case from the data shown here.**

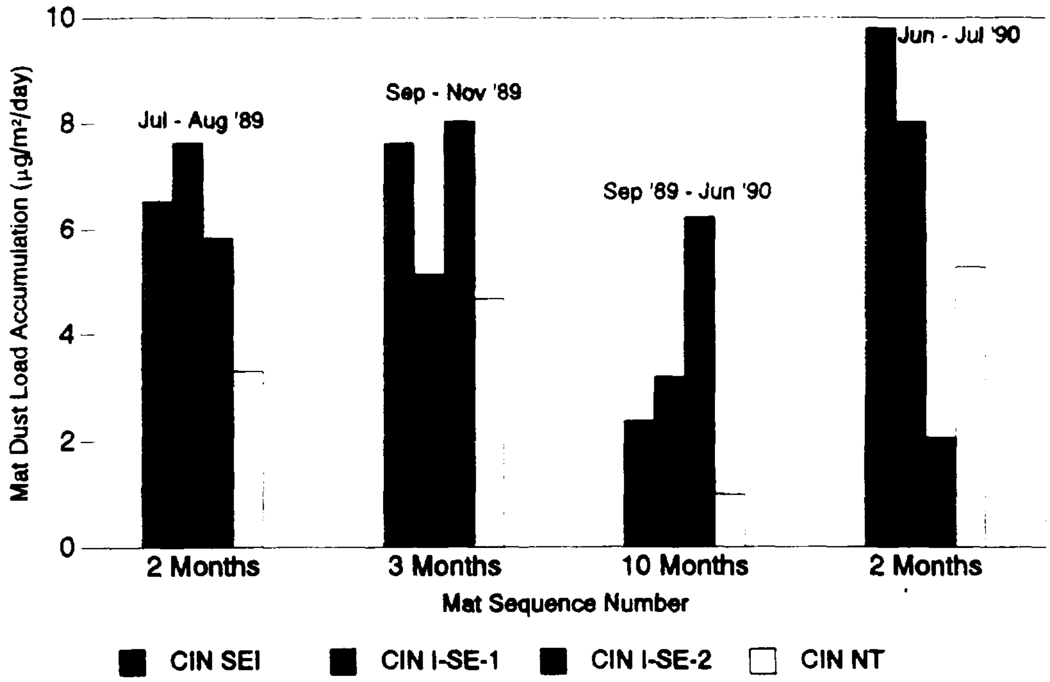


**Figure 3-32.** **Cincinnati mat dust lead load. These data appear to be consistent with the observation that there was an increase in exposure to house dust that occurred between November 1989 and October 1990.**

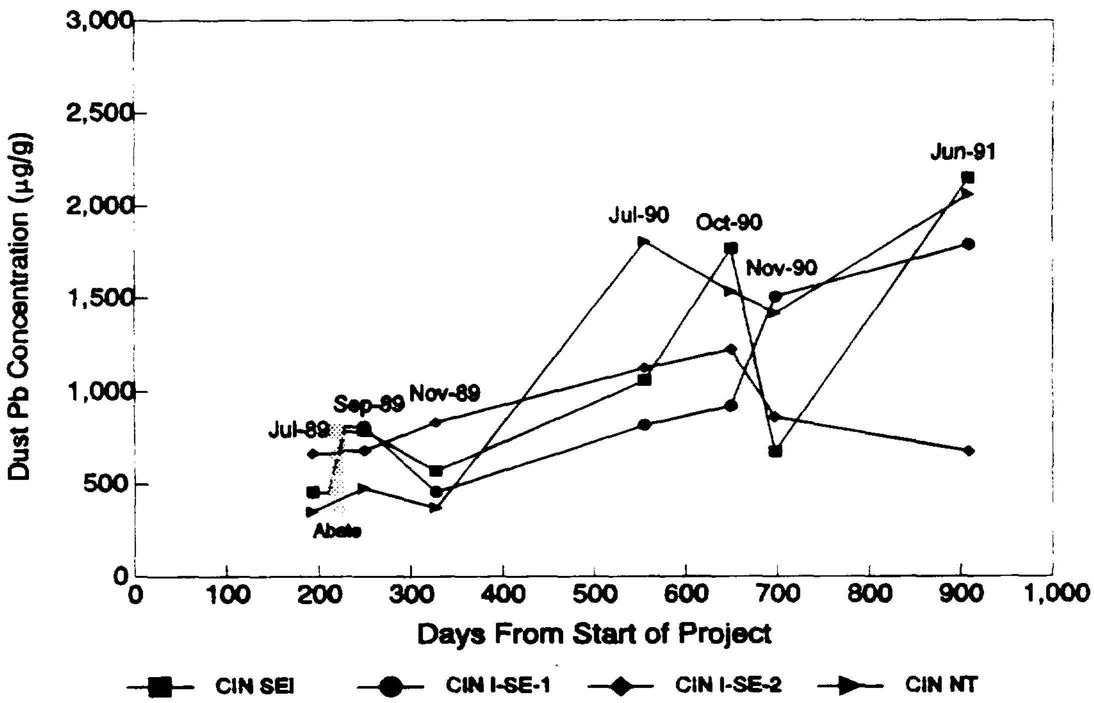


**Figure 3-33.** **Cincinnati entry dust lead concentration. The entry way subset of the floor dust shows a pattern different from the complete floor dust data of Figure 3-24 and the mat dust data of Figure 3-30. Note the two additional rounds, November 1990 and June 1991.**

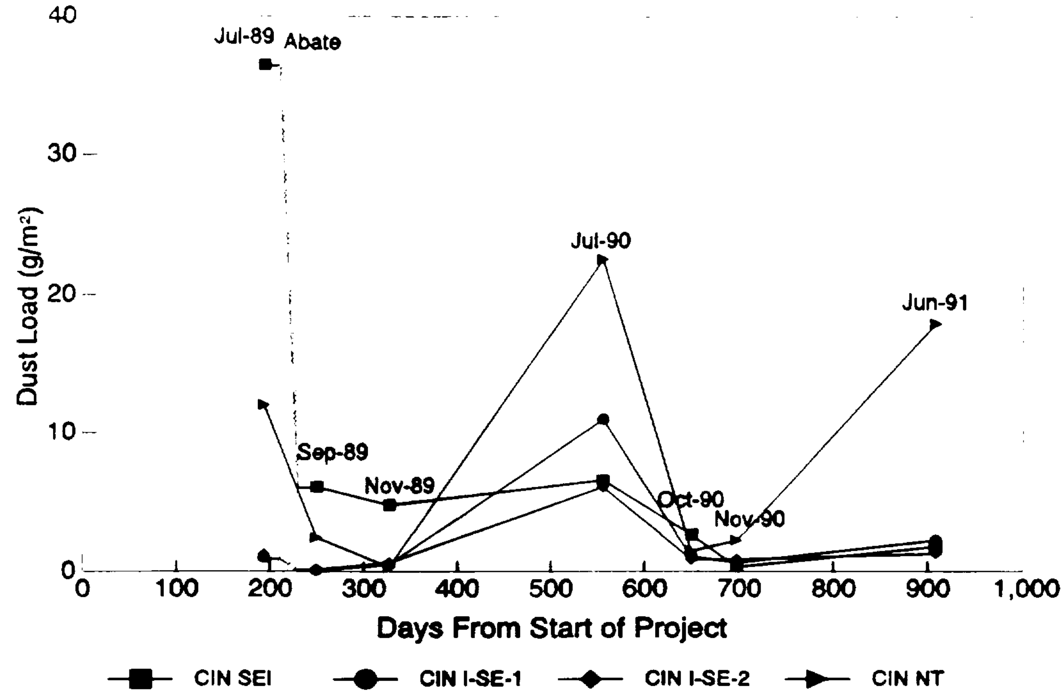


**Figure 3-34. Cincinnati entry dust load. Similar to Figure 3-25, dust abatement at the entry appears to have been effective and persistent through November 1989.**

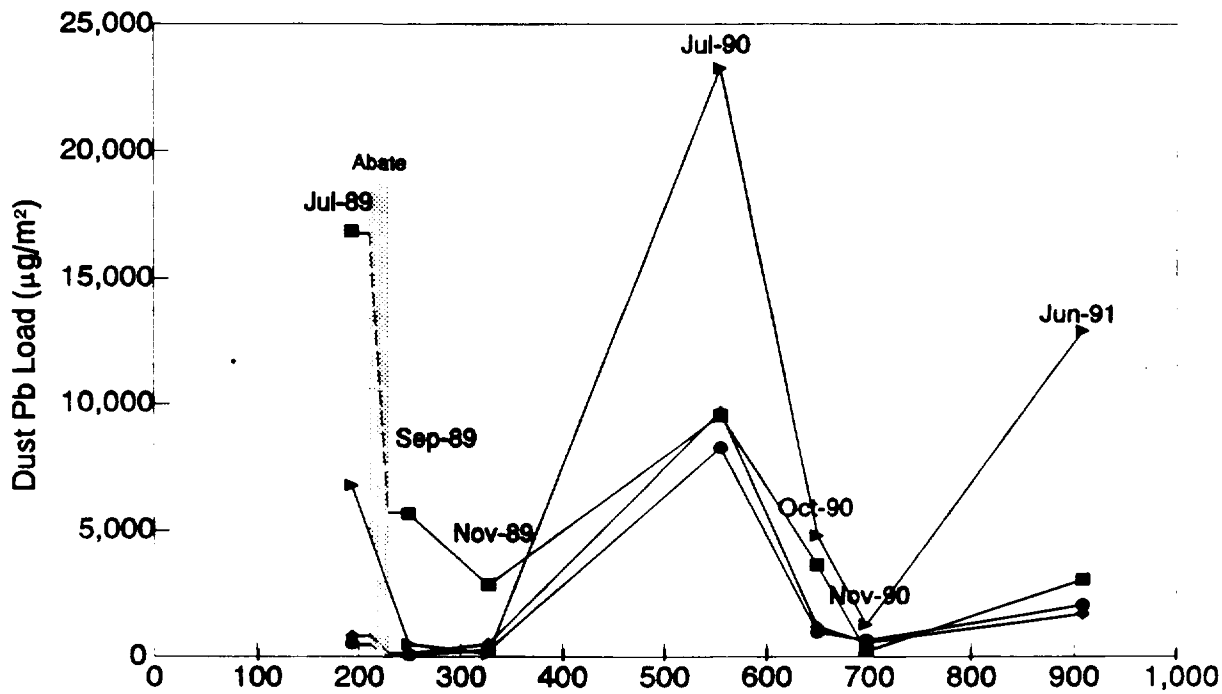


**Figure 3-35. Cincinnati entry dust lead load. There were a few housing units with entry dust lead loads three or more orders of magnitude higher than floor dust lead loads, which obscured the interpretation of the entry dust lead load.**

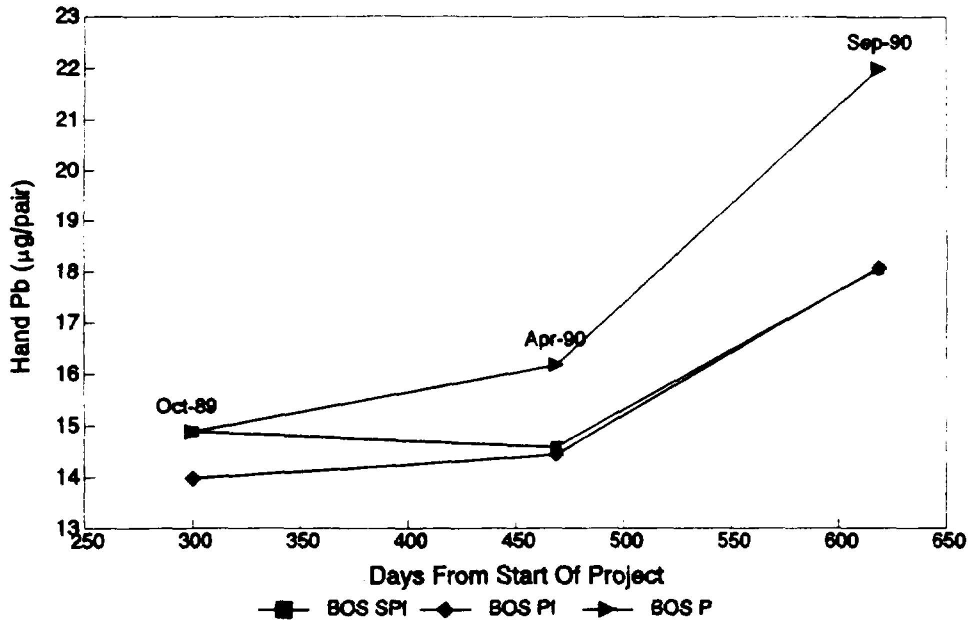


**Figure 3-36. Boston hand lead load. The amount of lead on the hands increased in the same manner as the floor dust load (Figure 3-19) and similar to the floor dust lead load (Figure 3-20).**

an effect for the groups that received soil and dust intervention, and this reduction is greatest for the group that received soil, dust, and paint intervention.

Baltimore hand lead values did not follow a discernable pattern (Figure 3-37) and there appears to be no difference between the two intervention groups.

In Cincinnati, the hand dust lead load (Figure 3-38) appears to follow the pattern of change observed in the floor dust lead load (Figure 3-26). This is an important link to establish in the exposure pathway. The measurement of lead on children's hands is a new exposure assessment tool that was introduced in this project as an alternative to blood lead measurements, which were expected to respond more slowly to environmental changes. Of the three studies, only the Cincinnati group had previous experience with hand lead analysis. The discussion below of the relationship of hand lead to blood lead will shed further light on this critical pathway.

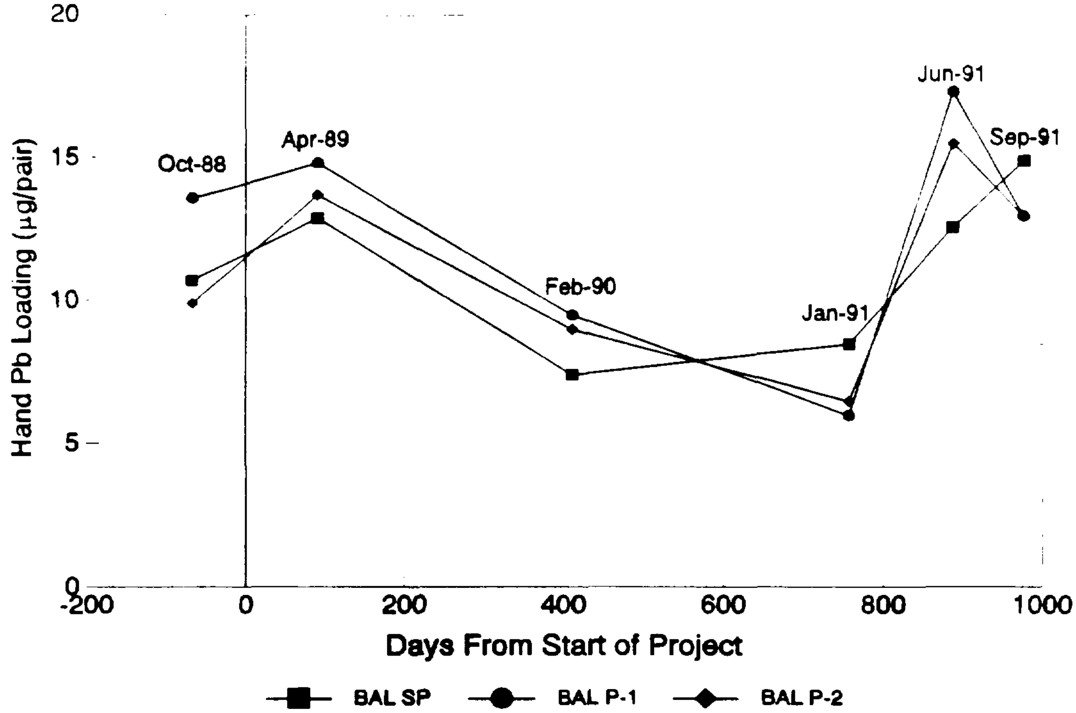


**Figure 3-37. Baltimore hand lead load. There were no sequential measurements of Baltimore house dust to compare the hand lead load.**

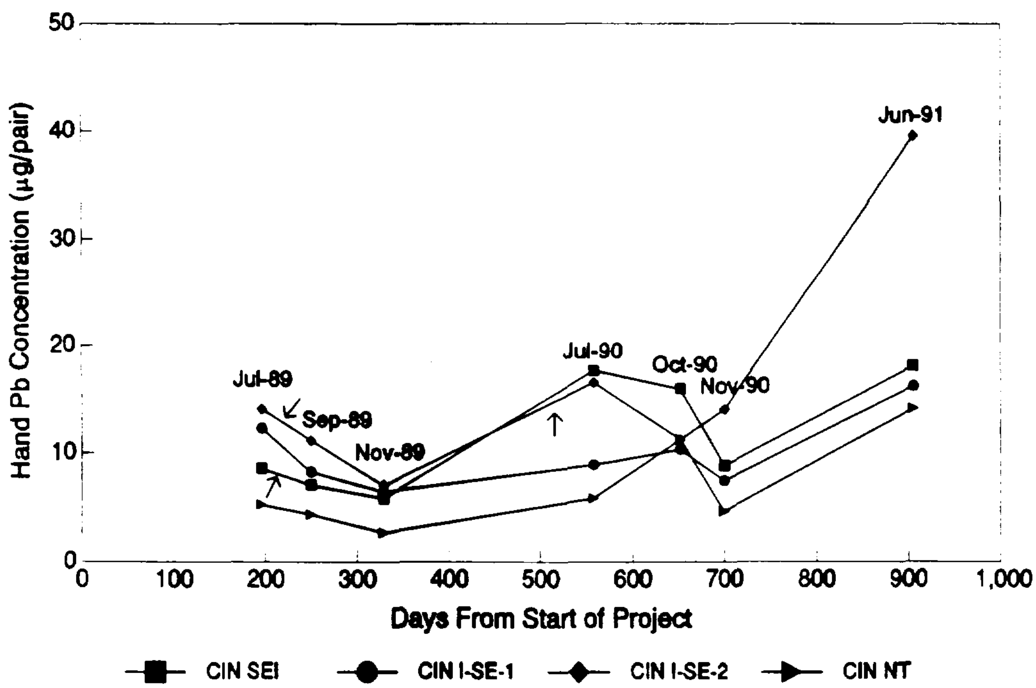


**Figure 3-38. Cincinnati hand lead load. The pattern of hand lead load change, both increases and decreases, appears to follow the pattern of floor dust lead load in Figure 3-26. Arrows indicate time of abatement.**

### 3.3.5 Blood Lead Results

The seasonal cyclic patterns and long-term trends observed in many studies of children's blood lead were discussed in Chapter 2. The importance of understanding these patterns is illustrated in the next series of graphs of the Baltimore data. The first, Figure 3-39, shows the uncorrected blood lead data that suggest the data are fairly smooth through most of the study, and essentially identical between groups. But there is a problematic increase at the end, especially for the BAL SP group. Notice that none of the rounds' measurements are taken at exactly the same time of year. Figure 3-40 fits a sinusoid to these data. The sinusoid has an amplitude of approximately 15% of the blood lead, a period of 1 year, and a maximum at August 18.

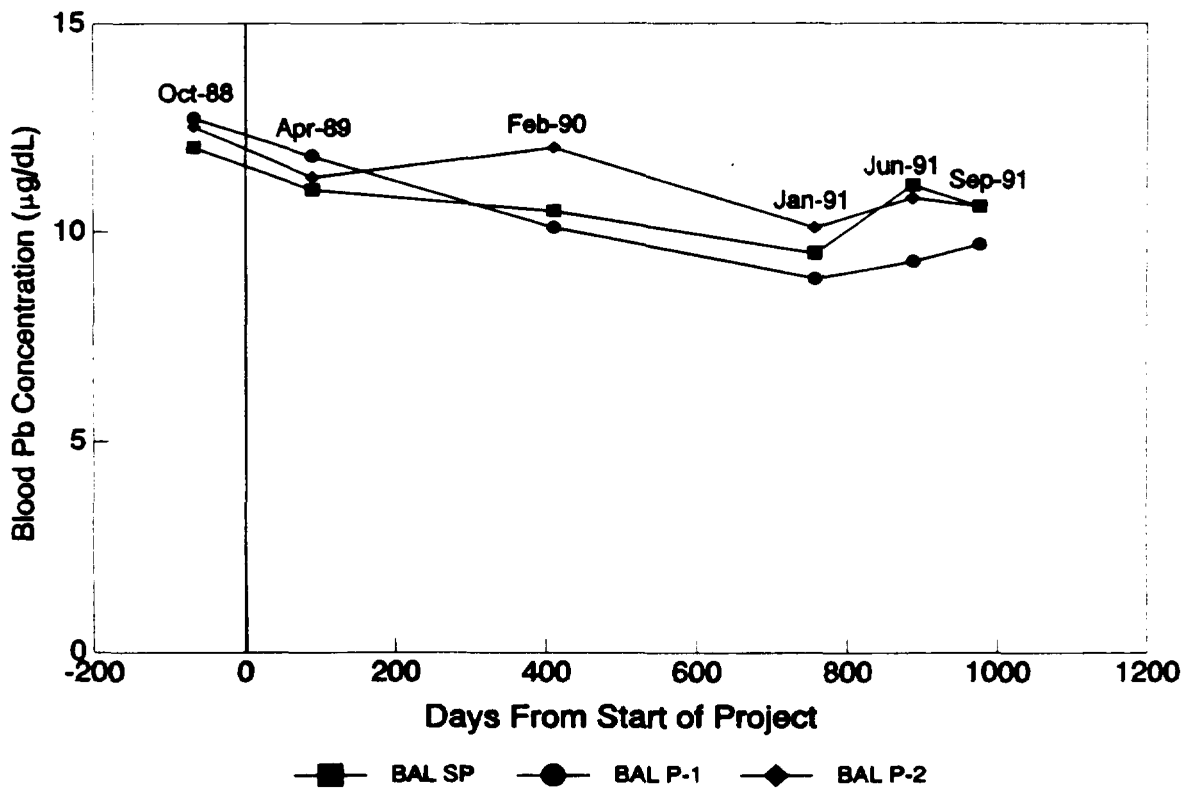


**Figure 3-39. Baltimore uncorrected blood lead concentrations. There appears to be little difference between study groups.**

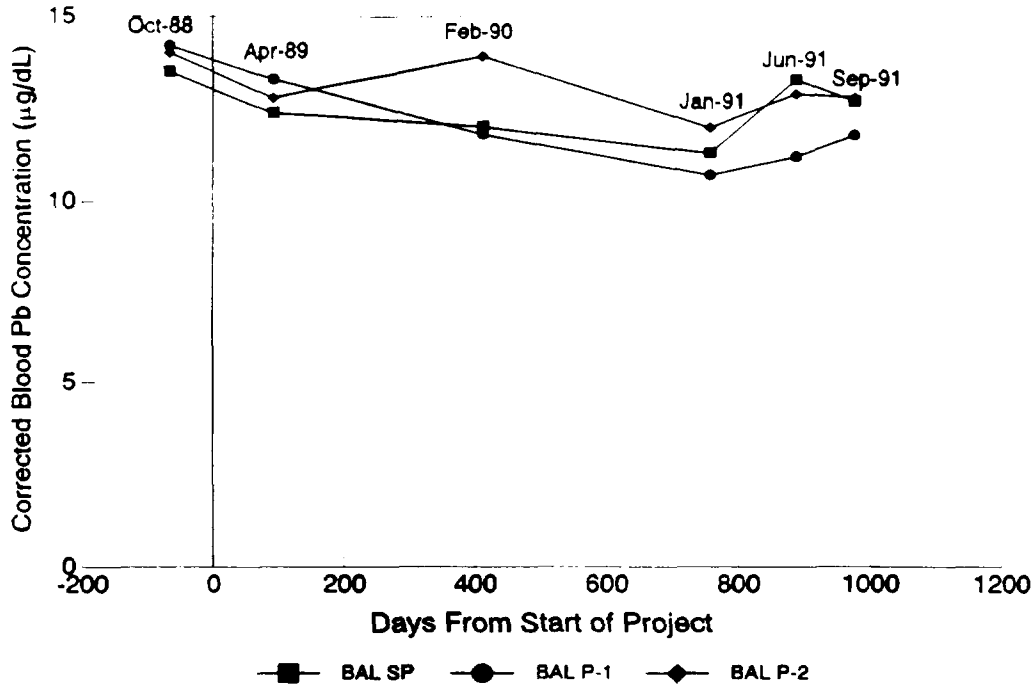


**Figure 3-40. Baltimore blood lead concentrations corrected for seasonal cycle and long-term time trends. The manner of correction for seasonal cycles is described in Figure 3-41.**

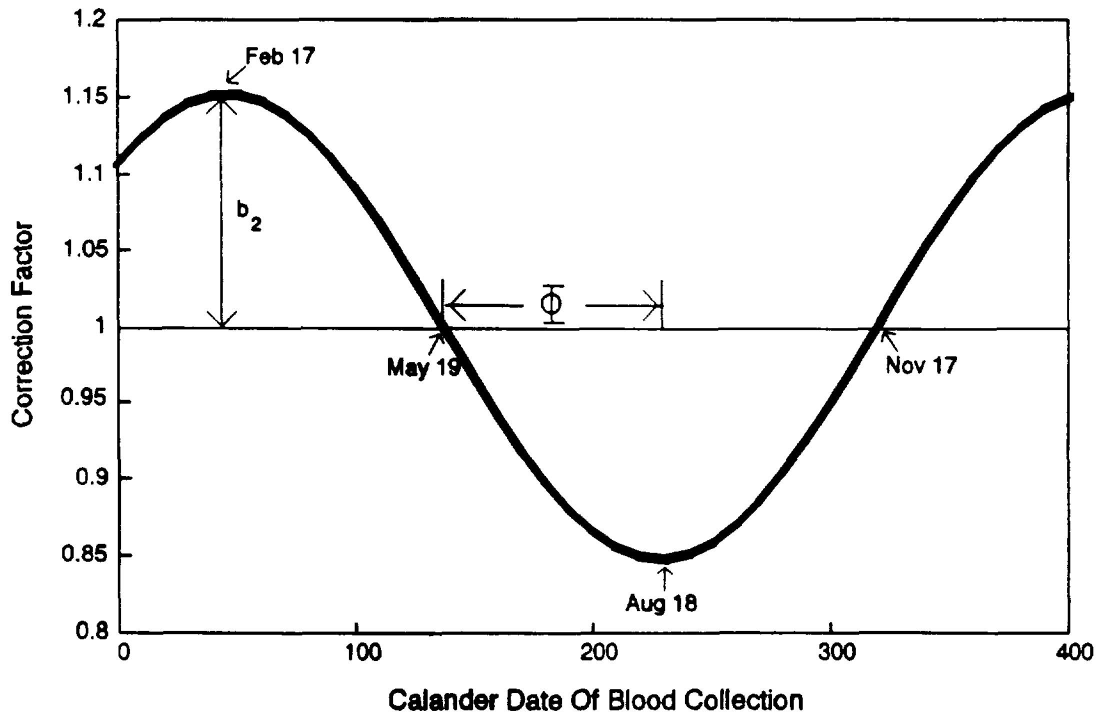


**Figure 3-41. Seasonally adjusted correction factor for blood lead concentrations. Blood lead concentrations typically vary by about 30% seasonally, and peak in the late summer. This correction appears is the same for all three studies, both in the offset (Φ) and the amplitude ($b_2$).**

#### 3.3.5.1 Blood Lead Correction Factors

By minimizing the impact of these seasonal effects, the impact of intervention becomes more systematic and more amenable to statistical evaluation and interpretation. The fact that the corrections made to the data were minimal, in the sense that the same peak date (August 18) and relative magnitude (15%) was applied to all children in all three studies argues strongly for the validity of this correction.

To correct for seasonal variations and apparent long-term trends in children's blood lead concentrations, the following equation was used:

$$CPbB_i = (PbB_i + b_1 d_i)\,[1 + b_2 cos(2\pi d_i/365 + \Phi)] \qquad (3\text{-}2)$$

where
- $CpbB_i$ = corrected blood Pb for child $i$, in $\mu$g/dL;
- $PbB_i$ = measured blood Pb for child $i$, in $\mu$g/dL;
- $b_1$ = time trend constant, in $\mu$g/dL/day;
- $d_i$ = date of blood measurement for child $i$, in days;
- $b_2$ = seasonal cycle constant, unitless;
- $\Phi$ = offset for calendar year, in radians.

The first segment of this equation, $(PbB_i + b_1 d_i)$, adjusts the measured blood lead for the long-term downward trend believed to exist for the general population. This constant is different for each city and can be measured in the child populations prior to intervention, or where no intervention occurred. The values for this constant were $-0.0025$ $\mu$g/dL/day for Boston and $-0.00025$ $\mu$g/dL/day for Baltimore and Cincinnati. The significance of the 10-fold difference between Boston and the other two studies will be discussed later.

The second segment, $(1 + b_2 cos(2\pi d_i/365 + \Phi))$, adjusts the blood lead for the seasonal cycle. This determination is based on a sinusoidal cycle and is largely independent of the observed blood lead concentrations. The two coefficients that impact this component of the equation are $b_2$ and $\Phi$, both of which are the same for all three studies. The seasonal cycle constant, $b_2$, determines the magnitude of the cycle, and the offset, $\Phi$, is based on the date of the maximum. The availability of three simultaneous longitudinal studies provided a rare opportunity to evaluate a large database for seasonal cyclic patterns. Analysis of the three studies led to the conclusion that the blood lead varied by ±15% throughout the year. Hence, the constant, $b_2$, is 0.15.

The peak occurred about August 18 for each study. The offset to the date midway between the maximum and minimum would be 91 days, or 0.82 radians in terms of the sinusoid. Figure 3-41 illustrates the manner in which the seasonal cycle correction was made.

### 3.3.5.2 Reanalysis of Boston Study Blood Lead Data

The uncorrected blood lead concentrations for the Boston study are shown in Figure 3-42, and the corrected values are shown in Figure 3-43. The corrected blood lead concentrations graphically illustrate the conclusions of the Boston report, that intervention probably accounted for a decrease of 0.8 to 1.5 μg/dL in the blood lead. The observation that all three Boston study groups experienced an increase in uncorrected blood lead concentrations between Round 3 (April 1990) and Round 4 (September 1990) is consistent with similar observations in the hand dust lead load and, to a lesser degree, the window dust lead load. The apparent absence of a comparable increase in floor dust lead load runs counter to the expected pattern of the floor dust lead load being the primary route for dust exposure in children.

### 3.3.5.3 Reanalysis of Cincinnati Study Blood Lead Data

The wealth of information from the more detailed measurements of household dust in the Cincinnati study presents a proportionally greater challenge to the paradigm of dust exposure pathways. The uncorrected blood lead concentrations shown in Figure 3-44 correspond roughly to the changes observed in the hand dust lead loads of Figure 3-38. And there are several points where the blood lead concentrations are consistent with the observed changes in the various forms of house dust. The floor and window dust lead loads are especially indicative to the exposure route, and the mat dust lead load seems to account for the increase in blood lead concentrations after November 1990.

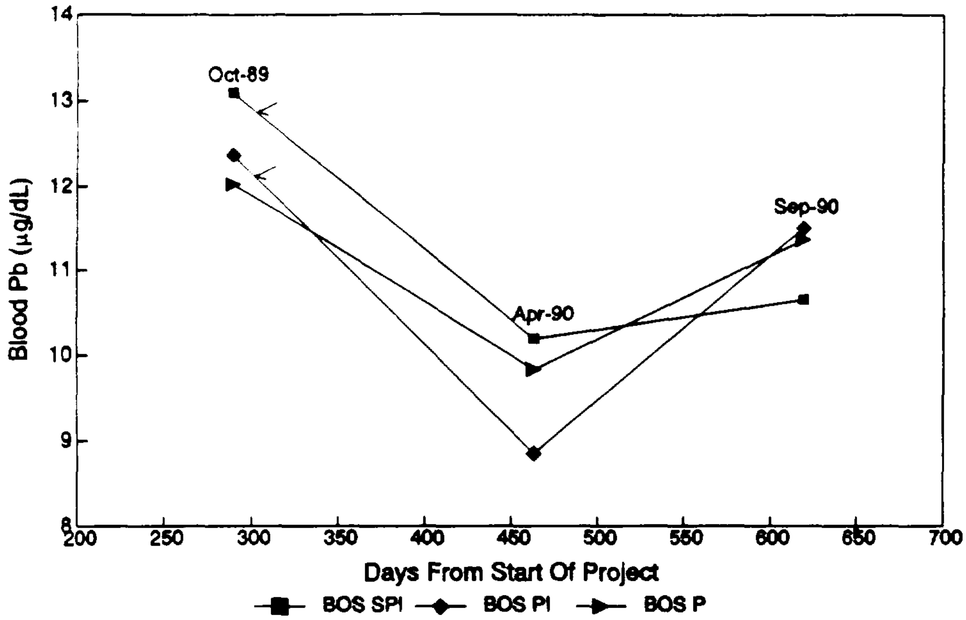


**Figure 3-42.** **Boston uncorrected blood lead concentrations. The approximate time of soil and dust abatement is shown by arrows.**

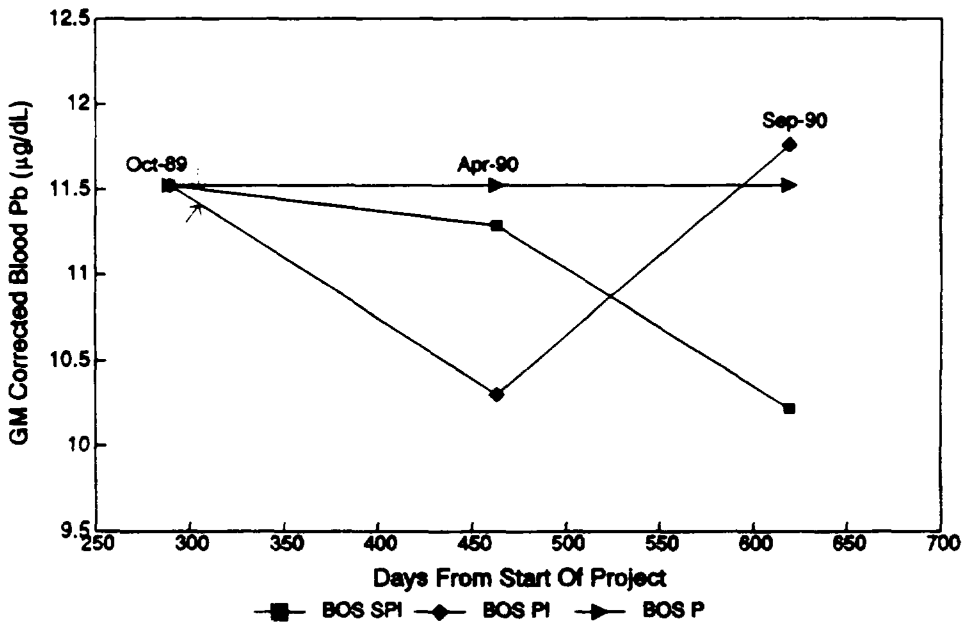


**Figure 3-43.** **Boston blood lead concentrations corrected for seasonal cycles and long-term time trends and normalized to BOS P. The approximate time of soil and dust abatement is shown by arrows.**

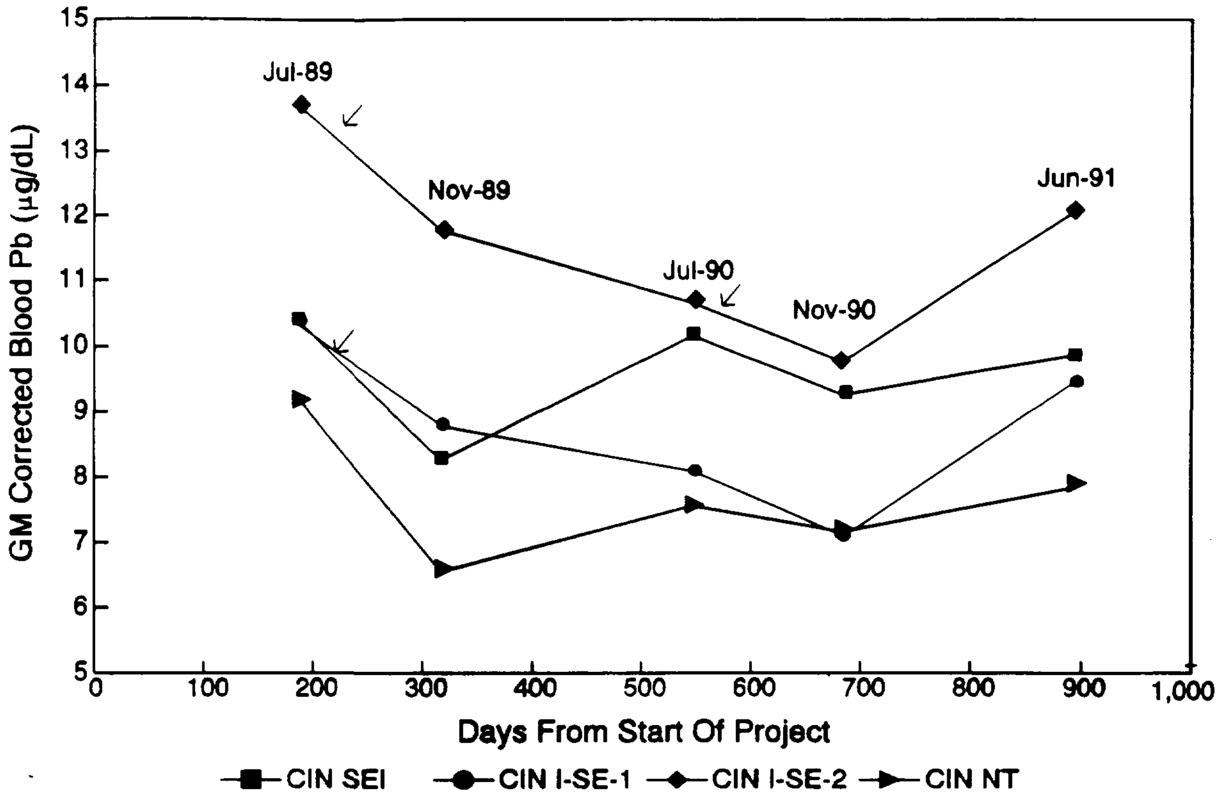


**Figure 3-44. Cincinnati uncorrected blood lead concentrations. The approximate time of soil and dust abatement is shown by small arrows. Compare to hand lead load patterns in Figure 3-38.**

The corrected blood lead concentrations in the Cincinnati study (Figure 3-45) suggest an impact of changes in environmental lead. On the same time scale as the Boston and Baltimore projects, the observed decrease at July 1990 (between 0.6 and 1.2 $\mu$g/dL) is similar to that seen in the Boston study. The group that received soil abatement in the first year, CIN SEI, continued to show increasing blood lead concentrations through the following year, and the CIN I-SE-1 and CIN I-SE-2 groups responded negatively following soil and exterior dust abatement in the second year. These uncertainties require that final conclusions on the impact of abatement be postponed until more detailed analyses, as described in Chapter 4, can be made.

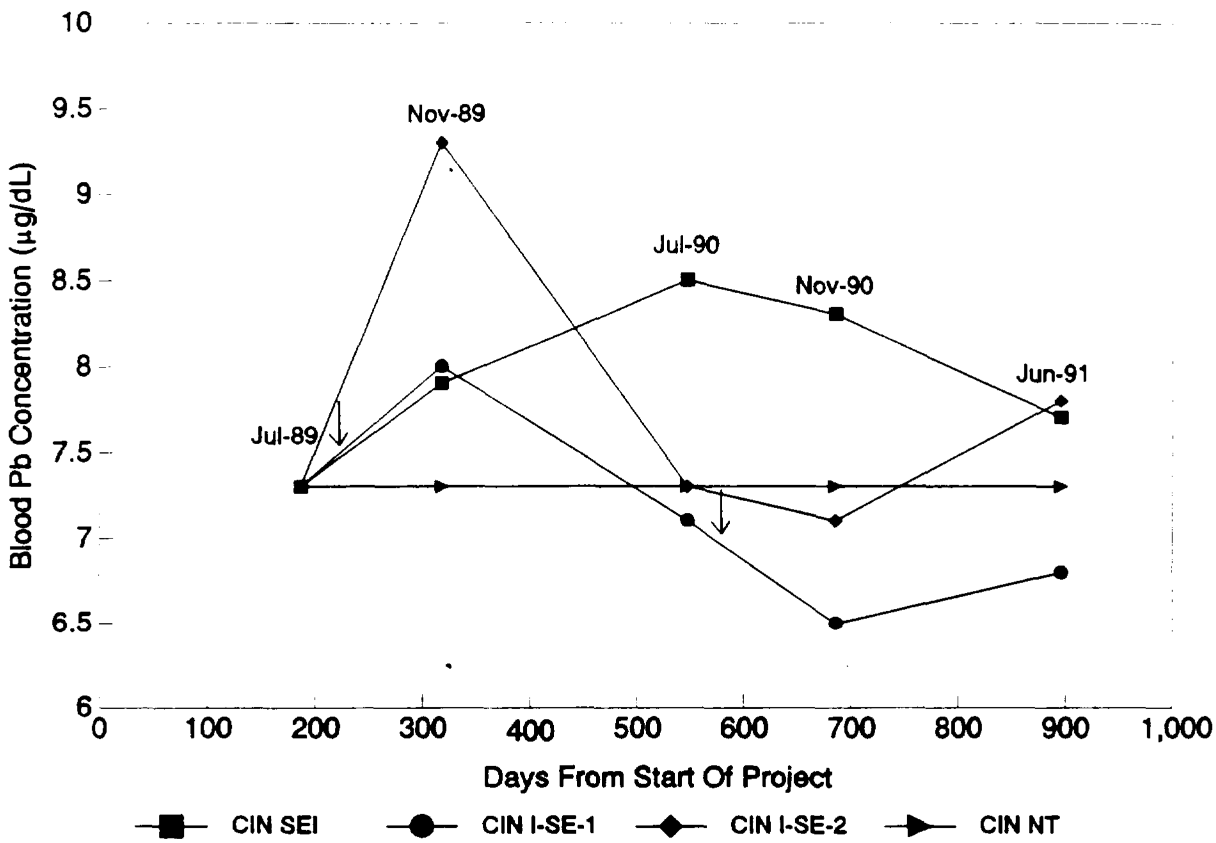


Figure 3-45. **Cincinnati blood lead concentrations corrected for seasonal cycles and long-term time trends and normalized to CIN NT to show possible impact of soil and dust abatement. The approximate time of soil and dust abatement is shown by small arrows.**

## 3.4 SUMMARY OF RESULTS

The data presented in this section lead to the following conclusions:

(1) Soil abatement in each study effectively reduced the concentration of lead in the soil in the areas where soil abatement was performed.

(2) In the Boston and Cincinnati studies, the effectiveness of soil abatement was persistent through the end of the study. There were no followup measurements of soil in Baltimore to determine persistency.

(3) Many of the study groups showed a slight downward trend in soil lead concentration independent of abatement.

(4) Exterior dust abatement, performed only in Cincinnati, was not persistent, indicating a source of lead other than soil at the neighborhood level.

(5) Interior dust following abatement as performed in Cincinnati and Boston, responded to subsequent changes in exterior dust and soil lead. Entry was measurements of lead concentration and lead load were a good indicator of the movement of environmental lead.

(6) Hand lead measurements reflected general trends in blood lead measurements and may be a reasonable estimate of recent exposure. However, the results do not shown that hand lead measurements, as performed in these studies, can be an adequate surrogate for blood lead measurements.

(7) Because paint stabilization was performed on all homes with lead-based paint in Boston (exterior and interior) and Baltimore (interior only), there is no measure of the effectiveness or persistency of this form of intervention.

From the standpoint of changes in environmental lead independent of intervention, there were few instances where changes in the blood lead concentrations could not be attributed to these changes. Some of these changes were expected, although uncontrolled. An example is the abrupt change in floor dust load between April and July 1990, shown on Figure 3-19. It appears that all homes were thoroughly cleaned during this period, but there was no corresponding decrease in the window dust load in Figure 3-22. Some changes were unexpected and equally difficult to explain. The dramatic increase in dust lead concentrations in the Cincinnati study between November 1989 and July 1990, observed in nearly every instance (floor, window, mat, and entry), remains unexplained. Although this apparent contamination appears to have overwhelmed the intervention efforts, the fact that both hand dust loads and blood lead concentrations responded to environmental lead gives credence to the strong link between environmental lead and blood lead.

In terms of changes attributed to intervention, it is appropriate to note that all three studies observed a quantifiable blood lead concentration change in response to soil, dust and paint intervention. The analyses in Chapter 4 will show that, although not always statistically significant, this quantifiable response to intervention is consistent even at low levels of environmental lead. Normalized to a decrease in soil lead concentration of 1,000 $\mu$g/g, the response in blood lead concentration appears to be about 1 $\mu$g/dL. This suggests that there is no plateau, within the ranges measured in this project, where the removal of environmental lead will not produce a corresponding reduction in blood lead concentrations.

# 4. STATISTICAL INTERPRETATION OF THE RESULTS

## 4.1 PREPARATION OF PROJECT DATA SETS FOR STATISTICAL ANALYSIS

Blood lead is a measure of the recent history of lead exposure and may respond to decreases environmental changes in lead within a 2- to 4-mo time frame. Reductions in exposure might be somewhat attenuated by the remobilization of lead in bone tissue. There is little information on these biokinetic translocations of lead when the total body burden is decreasing. If the total lead exposure of the child decreases, there seems to be no doubt that the blood lead concentrations would decrease, but there are no other comparable studies of the rate at which blood lead concentrations respond to decreasing lead exposure.

Although the objective of the project was to reduce blood lead concentrations in children by reducing the concentrations of lead in soil, other measures of the impact of soil abatement are possible and, indeed, perhaps more realistic. These are reductions in house dust lead concentrations and reductions in hand lead. The rationale for evaluating all three measures is that changes in house dust lead concentrations, especially in entry areas where the influence of exterior dust and soil is greatest, should be more responsive than blood lead to changes in soil lead and should not be influenced by nondust sources of lead, such as food and drinking water.

Hand dust reflects a mixture of dust lead sources throughout the child's environment, both indoors and outdoors. It may be a representative measure of total dust exposure, but the impact of soil abatement is diminished when there are sources of dust lead other than soil in the child's environment.

In this project, changes in blood lead must be interpreted in the context of four time-dependent effects that are independent of each other. These are

(1) the typical seasonal changes in children's blood lead concentrations, found in virtually every longitudinal study, that usually indicate a peak in concentration during the late summer months;

(2) the changes that occur with age during early childhood that usually peak between 18 and 27 mo;

(3) long-term changes in national baseline levels of exposure, believed to be mostly from reductions of lead in gasoline and in food, that are reflected in a downward trend for childhood blood lead levels observed since 1978; and

(4) changes that can be attributed to interventions of this project.

Evidence for all four of these trends and cycles can be extrapolated from the data, as discussed in Section 3.3.5, and this evidence is an important part of the statistical inferences drawn from the data.

### 4.1.1 Description of Data Sets

The data sets were provided by the principal investigators of the three studies. These data sets were edited to remove information that would identify the participants. Obvious data errors were detected and either corrected or eliminated, and data discrepancies were resolved by the Environmental Criteria and Assessment Office at Research Triangle Park, NC (ECAO/RTP). For each of the three studies, three files were created, as LOTUS (Lotus 1-2-3 v3.2, 1992) spreadsheets and then were exported into SYSTAT (Wilkinson, 1991) data files. The SYSTAT statistical system was used for additional data editing, including the creation of new variables in each data set, such as natural logarithms of lead variables and ages of the children when blood, hand, environmental, or interview samples were collected. Subsets of SYSTAT data files were used as input for structural equation modeling using EQS software (Bentler, 1989).

The three data files for each study were intended to be the child (KID), family (FAM), and property (PROP) files. The KID file was the unit that identified response to lead abatement. The FAM file was the unit that defined the interior dust and paint exposures and socio-demographic influences on lead exposure. The PROP file was the unit defined by the soil exposure and abatement data. In Cincinnati, the PROP file was subdivided into PROP and NBHD because most of the soil data could not be identified with a specific property.

The project data files were set up as

(1) a KID file with each child listed as a single record in the data set;

(2) a FAM file in which each family was listed as a single record in the data set, including those families with two or more siblings enrolled in the study;

(3) a PROP file in which each dwelling unit was listed as a single record in the data set; and

(4) a NHBD file (Cincinnati only) in which data from each soil parcel or exterior dust sampling location were listed as a single record.

### 4.1.2 Validation of the Data Sets

The data sets were validated by reproducing the statistical results reported by the three studies. Only minor modifications were made in the original data sets for this exercise. In all instances, the validation exercise reproduced identical numbers of participants.

Validations of the six models examined in the Baltimore report found no major discrepancies. The reported and validation results agreed for the regression coefficients and their standard errors.

Results for the appropriate hypotheses tests coincide with those in the Boston report. Analyses showed the average declines in blood lead levels between Round 1 and Round 2 (before and after abatement) to be significantly different from zero for all three groups. This pattern held throughout the validation of the Boston analyses.

### 4.1.3 Modifications in the Project Data Sets

#### 4.1.3.1 Restructured Data Sets

Certain modifications in the structure of the project data sets were necessary to facilitate the statistical analysis described below. Some households consisted of children from at least two distinct families, and children from each family were in the study, so that there were duplicates of dwelling unit data in some of the family file cases. There were also several cases in which there were two or more apartments in the same building. These were identified as separate premises in the Boston and Cincinnati studies, but could not be distinguished in the data set provided to ECAO/RTP by the Baltimore study group, and so were condensed to single premises. Most of the analyses reported here were performed using the KID data sets.

In Baltimore, soil lead was collected once at each dwelling unit near the beginning of the study, but only at the soil-abated dwelling units in BAL SP after abatement, which occurred between blood lead Rounds 3 and 4. Dust lead was collected only once, before abatement. In Boston, soil lead was collected once near the beginning of the study for all dwelling units and twice after soil abatement to study recontamination. Soil was collected immediately after abatement at Round 2 only for dwelling units in BOS SPI, where soil abatement had been performed. Dust was collected at Rounds 1, 3, and 4 for all dwelling units, but at Round 2 only for the dwelling units in BOS SPI and BOS PI, and not for BOS P. This introduced some built-in missing value patterns in the longitudinal design of the study.

#### 4.1.3.2 Missing Information Procedures

The Baltimore study design called for six rounds of blood lead measurements. Because only those children with all six measurements were used in the data analysis, a problem arose with the large number of children who were excluded from the analysis. To explore solutions to this problem, a method, called the Missing Information Principle, was used to fill in the missing data and analyze the entire data set. The method was described by Woodbury and Hasselblad (1970), and further refinements were made by Orchard and Woodbury (1972) and Dempster et al. (1977). The method is now called the E-M algorithm. It starts with any reasonable first estimate of the parameters followed by two steps, called the E and M steps. The E step consists of estimating the sufficient statistics, in this case the sums and sums of squares of cross products. The M step consists of recomputing the estimates of the mean and covariance from the completed sums and sums of squares and cross products.

For this analysis, the data set was limited to individuals present for the first round of measurements, but the procedure could be expanded with additional effort. The two assumptions for this analysis were that the blood lead data were missing at random and that the blood lead values were multivariate (log)normal. All observations were transformed logarithmically. Using the E-M algorithm, 20 children were returned to the data set.

A reanalysis of the data showed that Round 1 data are less correlated with the other five rounds than the other five are with themselves. For logistical reasons, this reanalysis was performed on blood lead data not corrected for seasonal cyclic patterns or long-term time trends. When Rounds 2 and 3 were used as the preabatement blood lead concentration and Rounds 4, 5, and 6 were used as the postabatement blood lead concentration, several observations emerged. First, the change in the group of children in BAL SP that did not receive abatement was not zero. This is consistent with the observation that there were seasonal cyclic patterns and long-term time trends in the blood lead data from all three studies. Second, the estimated change in the blood lead for BAL SP where abatement did occur was 1.1 $\mu$g/dL per decrease of 1,000 $\mu$g/g lead in soil. Although not statistically significant, this result is consistent with the results of the Boston study.

The imputing of missing values in the Baltimore data set was an exercise to see if additional information could be extracted using this technique. Time constraints prevented further statistical analyses that might refine the conclusions. For now, this procedure is treated only as evidence that the impact of intervention is continuous down to low levels of soil lead concentrations, although this impact may be too small to measure below a decrease of 1,000 $\mu$g/g.

## 4.1.4 Statistical Methods

### 4.1.4.1 Repeated Measures Analysis

The common form of the statistical analysis that can be most easily applied to data from all three studies is the form of multivariate analysis of variance or analysis of covariance called repeated measures analysis. In this method, observations on each subject or unit of the study are supposed to occur in an ordered sequence, normally a time series. The subjects are in separate treatment groups or categories that do not change with time. The response variable $Y$ (e.g., log blood Pb) for subject $i$ in group $j$ at time $t$ is denoted by $Y_{ijt}$. The model is then

$$Y_{ijt} = M + S_{ij} + G_j + R_t + A_{jt} + E_{ijt} \tag{4-1}$$

where

$M$ is the grand mean over all groups and times.
$S$ is the variability specific to subject $i$ in group $j$ for all times.
$G$ is the effect of being in group $j$ for all times,
$R$ is the effect of being observed at time or round $t$,
$A$ is the effect of being in group $j$ at time $t$ over and above the effect of being in group $j$ (e.g., abated versus not abated), and
$E$ is the random measurement error.

The effect $S$ is assumed to be random, and the effects $G$, $R$, and $A$ are "fixed" effects in that they are not expected to change over time. They are characteristics of the experimental design (i.e., neighborhood and treatment). The average fixed effect is zero. Using an asterisk (*) to denote an appropriate weighted average over the subscript, the assumptions are

$G_* = 0$ (average across all groups),
$R_* = 0$ (average across all rounds), and
$A_{j*} = A_{*t} = 0$ for the null hypothesis (average across groups, average across rounds).

The expected subject effect and the expected measurement error are zero. The sum of squares for testing the hypothesis that $G = 0$ (no treatment group effect) is that of the between-subject variability $S$, whereas the sum of squares for testing that $R = 0$ (no time effect, including significant linear trends or seasonal differences) or that $A = 0$ (there is no interaction between treatment category and time) is that of the within-subject or repeated measurement variability $E$. Parameter estimation of effects and tests of hypotheses were performed using the repeated measures option in the procedure Multivariate General Linear Hypothesis (MGLH) in the SYSTAT package (Wilkinson, 1991).

We were most interested in testing the null hypothesis that the interaction term $A = 0$, because the treatment or abatement occurs during the course of the study. Even if the treatment groups were equal before abatement, they are not expected to be equal after abatement.

A similar model can be used when there are numeric covariates available (here denoted generically $X_{ij}$ in Equation 4-2). In the standard repeated measures model, the covariates may be different for each subject, but pertain to the whole study and should not change with time. An alternative specification for use with time-dependent covariates is shown in the next section. The general form of the repeated measures analysis of covariance model is

$$Y_{ijt} = M + S_{ij} + G_j + R_t + A_{jt} + b_t X_{ij} + E_{ijt} \tag{4-2}$$

where

$b_t$ is the response coefficient (slope), and

$X_{ij}$ is the coviariate (e.g., log soil Pb).

The hypothesis tests for regression coefficients are similar to the tests for between-subject and within-subject variation.

A problem arises if the response variable $Y$ must be transformed, say by a logarithmic transformation for blood lead or for hand lead, in order to reduce skewness and to stabilize variances across treatment groups. The implied model for the original untransformed variable is then multiplicative in treatment effects and random variation. This is probably acceptable for the analysis of variance (ANOVA) model in Equation 4-1, but is likely to produce a physically or biologically meaningless specification for the covariate model in Equation 4-2 when the covariates are indicators of distinct and additive sources of lead, such as soil lead and interior lead-based paint. For this reason, we also evaluated an autoregressive regression model specification as an alternative approach to dealing with the longitudinal design of the study.

#### 4.1.4.2 Autoregressive Regression Models

***Biokinetic Basis for an Autoregressive Blood Lead Model***

Lead in blood is stored in various body tissues and organs for varying amounts of time. The red blood cells retain lead for a few days, the soft tissues such as the kidney retain lead for some weeks or months, and the skeletal tissues may retain lead for years or even decades. We will not consider multicompartmental models for blood lead in this report, except to note that the apparent blood lead half-life or the whole-body lead retention time may appear to depend on the interval between blood lead samples. The blood lead concentration (denoted *PbB*, measured in micrograms of lead per deciliter of whole blood) will depend dynamically on the absorbed lead uptake rate (denoted *U*, measured in micrograms of lead per deciliter of whole blood per day) and on the mean blood lead residence time (denoted *T*, measured in days). A first-order differential equation specification may be assumed as a reasonable approximation for low to moderate levels of exposure:

$$\frac{dPbB}{dt} = U - \frac{PbB}{T} \tag{4-3}$$

Equation 4-3 may be solved explicitly for constant intake rate, *U*:

$$PbB(t) = PbB(0)\ e^{-t/T} + UT\left[1 - e^{-t/T}\right] \tag{4-4}$$

Equation 4-4 has an important implication for models of blood lead observed over time. The present blood lead, *PbB(t)*, depends on the preceding blood lead, *PbB*(0), but the proportionality is not constant. In fact, the relationship is decreasing with increasing time interval *t*. Thus, *it is not appropriate to use the difference in blood lead levels between successive sampling times as an index of effectiveness of abatement*. In fact, Equation 4-4 implies that

$$PbB(t) - PbB(0) = [UT - PbB(0)]\,[1 - \exp(-t/T)] \tag{4-5}$$

Thus, the change in blood lead over time, for short periods of time, must depend on the starting blood lead *PbB*(0). The actual dependence is more complicated because lead has multicompartmental biokinetics instead of the one-compartment kinetics of Equation 4-3. After a long period of time, the information in starting blood lead *PbB*(0) is negligible and the current blood lead *PbB(t)* reflects only lead uptake *U*, which is approximately a linear function of the environmental lead levels.

***Fitting an Alternative Autoregressive Regression Model***

The model implied by Equation 4-4 can be estimated using nonlinear regression techniques. If we let *PbB*, *PbH*, *PbS*, *PbD*, and *PbP*, denote levels of lead in blood, hands, soil, dust, and paint, respectively, then a generic form of an autoregressive regression model is

$$PbB(t) = PbB(t\text{-}1)\ r(t) + (b_1(t) + PbH(t) + b_2(t)\ PbS(t) + b_3(t)\ PbD(t) + b_4(t)\ PbP(t) + \ldots \qquad (4\text{-}6)$$

As noted above, log-transforming blood lead to reduce skewness and stabilize variances requires another specification for model parameter estimation:

$$\log[PbB(t)] = \log[PbB(t\text{-}1)\ r(t) + b_1(t)\ PbH(t) + b_2(t)\ PbS(t) + b_3(t)\ PbD(t) + b_4(t)\ PbP(t) + \ldots] \qquad (4\text{-}7)$$

This was fitted using the procedure NONLIN in the SYSTAT package (Wilkinson, 1991). The models fitted here show that there is a dose-response of blood lead to environmental lead. The effectiveness of abatement is thus reduced to testing the effectiveness of the abatement in reducing environmental lead. If the postabatement regression coefficients, $b(t)$, are not significant, then there is no persistent effect of abatement that is not characterized by the autoregressive coefficient, $r(t)$. Even if $b(t)$ is not significant, but $r(t)$ for the abatement group is significantly smaller than $r(t)$ for the nonabatement group, then there is a persistent long-term effect of abatement.

#### 4.1.4.3 Structural Equations Modeling

The most complete and technically correct evaluation of these studies requires a simultaneous assessment of changes in blood lead levels and changes in environmental lead pathways following soil lead and/or dust lead abatement. Underlying any analysis of time-dependent relationships are the following assumptions:

(1) Both preabatement and postabatement blood lead levels reflect, in part, contemporary environmental lead exposures that can be characterized by measurements of lead levels in soil, dust, paint and other media;

(2) Postabatement blood lead levels may also reflect, in part, preabatement blood lead levels due to the contribution of preabatement body burdens of lead (principally in the skeleton) from earlier exposures;

(3) Postabatement dust lead levels may also reflect, in part, preabatement dust lead levels due to mixing of incompletely abated or unidentified sources of lead in dust for which preabatement dust lead levels are a surrogate indicator;

(4) Postabatement soil lead levels may also reflect, in part, preabatement soil lead levels due to mixing of incompletely abated or unidentified sources of lead in soil for which preabatement soil lead levels are a surrogate indicator;

(5) Even when lead-based paint has been stabilized, lead paint levels measured by XRF may also help to predict postabatement soil and dust lead levels from incompletely abated or unidentified sources of lead in soil and dust for which lead-based paint levels are a surrogate indicator.

Based on these assumptions, there are several testable hypotheses that can be formulated:

HYPOTHESIS 1. Postabatement blood lead levels in the abatement group(s) are relatively lower than blood lead levels in nonabatement or low-impact abatement group(s), after adjustment for current environmental lead exposures, due to a persistent effect of the abatement in reducing body burden;

HYPOTHESIS 2. Postabatement blood lead levels in the abatement group(s) are not necessarily lower than blood lead levels in nonabatement or low-impact abatement group(s), after adjustment for current environmental lead exposures, but are lower in the abatement groups for which there has been a persistent reduction in soil and dust lead exposure levels.

An explicit parametric model for these hypotheses is based on the following relationship between preabatement and postabatement blood lead levels for each subject:

$$\text{PbBlood(Post)} = R * \text{PbBlood(Pre)} + B_0 + B_1 * \text{PbSoil(Post)} + \ldots \qquad (4\text{-}1)$$

The autoregression coefficient R in Equation A includes such factors as the exponential washout or biologic elimination of lead from the body over time. R also includes other time effects such as seasonal variations and a general reduction in lead exposure from diet and airborne sources. The valid use of Equation A requires that pre- and postabatement measurements be as nearly synchronous as possible. *All three studies have preabatement and postabatement blood lead measurements taken during the summer or early fall, about 10 to 12 mo apart. These can be used as a basis for comparison among the three studies, without requiring substantial adjustments for seasonality and time trends.*

A more complete analysis of these data would also use the measurements made immediately after abatement, and longer-term measurements assessing the extent and response to environmental recontamination, and aging of the subjects. While time series models are usually expressed in terms of autoregression of residuals, the lack of synchrony between blood lead samples and environmental samples makes such analyses less useful in the present situation.

The models shown here represent the best models we obtained by backward elimination of nonsignificant predictor variables from the most complete model, including those predictor variables for blood lead that were predicted pathway variables. Data were analyzed using the EQS program (Bentler, 1991) with GLS (generalized least squares) or AGLS (asymptotically distribution-free generalized least squares) methods. Cases with missing values of model variables were eliminated, and no effort was made at this time to impute any of the missing values. We tested model specifications with combinations of dust lead concentrations and loadings. All of the lead pathway equations may be developed by writing down linear models implied by the pathway diagrams. Hand lead was not used at this time, due to the complexity of the longitudinal models.

Negative coefficients in the model are physically or biologically uninterpretable, and coefficients were constrained to be nonnegative. Most of the zero-constrained coefficients were associated with variables eliminated from the model, and the remaining zero-constrained coefficients should not seriously affect the predictions relative to a nonconstrained set of coefficients. However, since all of the coefficients were constrained to be nonnegative, *one-tailed statistical tests should be used in interpreting the results.*

### 4.1.5 Limitations of the Statistical Methods

The statistical methods we used here were reasonably appropriate and could be used by many other investigators with access to standard statistical software packages. However, the methods have certain limitations that should be understood. The repeated measures analyses assume only that the response variables are correlated with each other, with no implication of temporal causality. The goodness of fit of the models was significantly improved by use of covariate analyses. The usual repeated measures analyses require that the covariates have no time dependence. This is appropriate for exposure covariates such as lead-based paint levels, which were not changed during the course of the study. It could also be used for dust lead levels in the Baltimore study that were only measured at one time, although there is a high likelihood that the dust lead levels were changing during the course of the study. The availability of environmental data to characterize time-varying lead exposures in the Boston and Cincinnati studies suggests that more powerful statistical methods, such as structural equation models, would be more appropriate.

It is also important to understand that the repeated measures model applied to log(Blood lead) implies that blood lead levels are multiplicative functions of their main factors. By using a response variable,

$$Y = \log(\mathit{Blood\ lead})$$

and exponentiating Equation 4-1 or Equation 4-2, we obtain

$$\mathit{Blood\ lead}_{ijt} = e^{M} \times e^{S_i} \times e^{G_j} \times e^{R_t} \times e^{A_{jt}} \times e^{E_{ijt}} \qquad (4\text{-}8)$$

In this model, $e^{M}$ is the geometric mean blood lead over all groups, treatments, rounds, and subjects. For the covariate model in Equation 4-2, if we used

$$X_{ij} = \log(\mathit{Environmental\ lead})_{ij}$$

as we did in Chapter 3, and exponentiate Equation 4-2, we obtain

$$Blood\ lead_{ijt} = e^{M} \times e^{S_i} \times e^{C} \times e^{R} \times e^{A_i} \times (EnvironmentalPb)_{ijt}\, e^{E_i} \qquad (4\text{-}9)$$

This model does not reproduce the additive model we believe is more appropriate.

The autoregressive regression model is closer to the basic biokinetics of lead exposure. However, the model implied by Equation 4-4 is only partially correct since the exposure variables embraced in the $U$ term are also varying over time. There are secular trends, such as steadily decreasing exposures from air lead and from lead in food cans. There are seasonal variations that may be multiplicative in nature. It would be preferable to fit a parametric form of the model. For example, to include a multiplicative seasonal variation with relative amplitude denoted $a$ and phase angle denoted $F$, we would use

$$\begin{aligned} \log[PbB(t)] = \log[&PbB(t\text{-}1)\, r(t) + b_1(t)\, PbH(t) + \\ &b_2(t)\, PbS(t) + b_3(t)\, PbD(t) + b_4(t)\, PbP(t) + \ldots] + \\ &\log[1 + a \times \cos(t/58.131 + F)] \end{aligned} \qquad (4\text{-}10)$$

We have used 58.131 as the number of days in a year of 365.25 days that are equivalent to a radian of angle. When time-varying covariates are not available for important predictors, such as the lack of hand lead levels in the data set for Cincinnati that was provided to us, then it may be more informative to use a model in which the relative reduction in blood leads postabatement could be used in the form

$$\log[PbB_{ij}(t)] = \log[PbB_{ij}(t-1)\, r_j(t) + b_1(t)\, PbD_{ij}(t) + \ldots] \qquad (4\text{-}11)$$

where the autoregression coefficients $r_j(t)$ may depend on both group identifier $j$ and time (round) $t$. The model in Equation 4-11 provided a better fit than the covariate-adjusted group means autoregressive model,

$$\log[PbB_{ij}(t)] = \log[PbB_{ij}(t-1)\, r(t) + g_j(t) + b_1(t)\, PbD_{ij}(t) + \ldots] \qquad (4\text{-}12)$$

where the intercept terms denoted $c_j(t)$ may depend on both group identifier $j$ and time (round) $t$, but the autoregression term $r(t)$ is the same for all groups.

Extension of repeated measures analyses to covariates such as environmental lead levels that change with time, and extension of the autoregressive regression models to simultaneous estimation of parameters at multiple time points where the output of one regression equation is used as input for the next, can be done using a single technique, structural equation modeling. These analyses are more powerful and general diagnostic tools.

## 4.2 IMPACT OF INTERVENTION

### 4.2.1 Impact of Soil Abatement on Exterior and Interior Dust

Exterior dust was measured and abated in Cincinnati only. In this study, the results suggest a recontamination rate for exterior dust of less than 2 weeks, and that the source of this recontamination is not the soil. Additional measurements have been made to identify the source and rate of recontamination, but no data are available at this time. The source may be a single episode (possibly external), a seasonal effect such as dry weather, or a continuous process possibly related to traffic. With a neighborhood level perturbance of this type, it is not possible to measure the impact of soil abatement on house dust directly. However, if abatement is considered on the broader scope, where neighborhood cleanup would include soil, external dust, and any other sources of lead external to the home, then the house dust measurements made immediately inside the homes can be used as a measure of this "total neighborhood abatement". For those cases in the Cincinnati study where there was no immediate recontamination of this entryway dust, this measurement can be used as a surrogate for soil abatement. To make this determination, it is also necessary to evaluate the fraction of exposure that would derive directly from soil or from playground dust, which would not be included in the interpretation of house dust alone.

The key to understanding the impact of soil (and external dust) abatement on interior dust is to observe changes in the three components of the interior dust measurement: lead concentration (micrograms of lead per gram of dust), lead loading (micrograms of lead per square meter), and dust loading (milligrams of dust per square meter). Where there was no interior dust abatement, the lead concentration in interior dust should decrease gradually over time, provided that the influence of lead-based paint has been minimized. Also, the lead loading should decrease if the dust loading remains constant or the lead loading is normalized to dust loading. This normalization is believed to correct for differences in housekeeping efficiency. If interior dust abatement has occurred, the lead concentration should decrease markedly and remain low where the influence of lead-based paint is minimal, and the lead loading and dust loading should decrease and then increase in tandem.

The impact of lead-based paint can be minimized in three ways: (1) observe only cases where there is no lead-based paint; (2) stabilize the paint so that the rate of incorporation to house dust is minimized; and (3) compare measurements where the influence of lead-based paint is probably high, such as window wells, to areas where the influence of soil is high, such as entryways. A crude measure of the rate of recontamination of house dust from lead-based paint can be observed from the changes in window well dust lead concentrations following interior dust abatement, for units with and without lead-based paint.

The analysis of three types of internal dust measurements, (1) entry, (2) floor, and (3) window well, can provide additional information about the impact of soil abatement. The entry measurement probably shows the greatest influence of exterior lead from soil and dust. If the entryway to the housing unit is somewhat removed from the building entrance, such as an apartment on the second or third floor, then a comparison of these two measurements should demonstrate the effect of soil lead on multifamily houses. Likewise, where interior dust abatement has taken place, the rate of recontamination of interior dust should be entry > floor > window well.

### 4.2.2 Impact of Soil and Dust Abatement on Hand Lead Loading

It was expected that hand dust would serve as an surrogate measure of changes in exposure following abatement to augment information about blood lead changes. Hand dust reflects the child's recent exposure (since the latest hand washing), but is a measure only of lead loading, not lead concentration or dust loading, because the total amount of dust is not measured. Consequently, it is not possible to estimate the source of lead (soil or paint) by differences in concentration, nor is it possible to correct for housekeeping effectiveness by observing changes in dust loading, as with house dust. It is believed that the amount of dust (not mud or dirt) on the hand reached equilibrium after a short period of time, perhaps 30 min to 2 h. The dustiness of the house would affect only the rate at which this equilibrium is reached, not the total amount of dust at equilibrium.

The hand dust measurements in this report should be viewed with caution because of the analytical difficulties discussed earlier and in the individual study reports. Nevertheless, both the Boston and Cincinnati studies showed a reduction in lead loading on the hands following interior dust abatement, but very little response to soil abatement.

### 4.2.3 Impact of Soil and Dust Abatement on Blood Lead Concentrations

Blood lead concentrations should respond to soil and dust abatement through the impact of abatement on two routes of exposure: (1) hand-to-mouth activity, reflecting the impact of interior house dust and exterior play area dust on exposure; and (2) food contamination, reflecting the incorporation of house dust in food during kitchen preparation. There was no measure of the incorporation of house dust into food during this project. Intuitively, the impact of interior dust abatement should be the same, or at least comparable, for food and hand dust. In some homes, however, lead-based paint is more common in kitchens and bathrooms, and the rate of return of lead-based paint following stabilization would have a greater impact on food than hand dust. There is a limited amount of data, not yet analyzed, where kitchen floor dust can be compared to bedrooms and other living areas, and likewise for window wells. Most of these data, however, are from the Cincinnati study, where there was a minimum influence of lead-based paint.

The Boston study showed a small but statistically significant effect of soil abatement on blood lead. This is expressed as a 1 to 1.5 $\mu$g/dL decrease in blood lead per 1,000 $\mu$g/g decrease of lead in soil. Although a greater effect was expected, these findings are not surprising for two reasons. First, the earlier studies that predicted a greater effect were not based on a reduction in exposure but on extrapolations from data of cross-sectional studies of children with different ranges of exposure to soil lead. Second, the measurement of soil lead in these earlier studies was of a distinctly lower quality than in this project. Fewer samples were taken, little effort was made to take representative samples, and no reference materials were available to standardize analytical procedures. In these earlier studies and the Boston study, there was no attempt to measure the impact of neighborhood-level exposure.

The Baltimore study showed no influence of soil abatement on blood lead concentrations. The Baltimore study did not measure the impact of soil abatement in the absence of interior lead-based paint, and it is possible that soil abatement would be swamped by the presence of paint lead in the house dust. This negative result is an important finding of this study and the integrated project that suggests, in the absence of interior dust abatement and interior paint stabilization (or abatement), soil, exterior dust, and exterior paint abatement will have little impact on childhood lead exposure.

The Cincinnati study showed no effect of soil abatement alone on the blood lead concentrations, but showed a positive effect of interior dust abatement and a marginal effect of total abatement when the interior-entry dust immediately inside the home was used as a surrogate of neighborhood lead abatement. The importance of these findings is that when the sources of lead that recontaminate exterior dust can be identified and abated, the impact of neighborhood-level abatement will be greater than single dwelling abatement alone.

## 4.3 RESULTS OF STATISTICAL ANALYSES

### 4.3.1 Baltimore Study

The repeated measures analysis of variance is shown in Table 4-1. The main effects of category (Control in Area 1, Control in Area 2, Abatement in Area 2) are nonsignificant in Table 4-1 ($p = 0.18$), due to the large between-subjects variance MS = 1.17. There is a large difference within subjects among Rounds 1 through 6, with $p < 0.000001$. The round by category interaction (soil abatement effects) are very small, none exceeding 0.039 (4% difference in blood lead) and show no consistent pattern of pre-Round 3 versus post-Round 3 direction. The soil abatement effects appear to be random, and this is borne out by the results in Table 4-1, where $p = 0.8886 \gg 0.05$.

Attempts to refine the analysis by use of covariates is shown in Table 4-2. This reduces the category effects overall, with a corresponding nonsignificant $p = 0.86$ in Table 4-2. The initial interior dust lead loading is highly predictive of blood lead levels ($p = 0.0079$), and the interior lead-based paint level is still marginally significant even after interior dust loading is considered ($p = 0.0598$). However, the round by category interaction is still nonsignificant ($p = 0.59$), so that soil abatement apparently had little effect on blood lead levels.

TABLE 4-1. REPEATED MEASURES ANALYSIS OF VARIANCE FOR LOG (BLOOD LEAD) FOR BALTIMORE STUDY, ANALYSIS OF VARIANCE TABLES

| Effect | MS | df | p |
|---|---|---|---|
| Between Subjects | | | |
| Category | 2.0305 | 2 | 0.1809 |
| Variability | 1.1729 | 139 | |
| Within Subjects | | | |
| Round | 2.0587 | 5 | $< 10^{-6}$ |
| Round * Category | 0.0253 | 105 | 0.8886 |
| Variability | 0.0504 | 695 | |

TABLE 4-2. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR LOG (BLOOD LEAD) FOR BALTIMORE STUDY, ANCOVA TABLES

| Effect | MS | df | p |
|---|---|---|---|
| Between Subjects | | | |
| Category | 0.1579 | 2 | 0.8621 |
| Log (XRFE) | 1.5108 | 1 | 0.2358 |
| Log (XRFI) | 3.8463 | 1 | 0.0598 |
| Log (PbLD-AA) | 7.7800 | 1 | 0.0079 |
| Variability | 1.0629 | 108 | |
| Within Subjects | | | |
| Round | 0.2487 | 5 | 0.0001 |
| Round × Category | 0.0396 | 10 | 0.5926 |
| Round × Log (XRFE) | 0.0246 | 5 | 0.7613 |
| Round × Log (XRFI) | 0.0831 | 5 | 0.1201 |
| Round × Log (PbDL-AA) | 0.0222 | 5 | 0.7999 |
| Variability | 0.0473 | 540 | |

This hypothesis is further developed in Tables 4-3 and 4-4. In Table 4-3, there is a statistically significant relationship between hand lead and blood lead (one-tailed $p < 0.05$) for Rounds 1, 2, 4, and 6, and a marginally significant relationship (one-tailed $p < 0.10$) for Round 3. A more detailed examination in Table 4-4 shows that there is a significant relationship between blood lead and at least one of the variables (hand lead, dust lead, or interior paint lead) preabatement at Rounds 1, 2, and 3. Table 4-5 shows that this also occurs in Round 4 for both control and soil abatement groups, and in Round 6 for the soil abatement group, but not in Round 5 or in the Round 6 control group. Soil lead is a significant predictor of blood lead in Round 2, and in the soil abatement group at Rounds 4 and 6.

**TABLE 4-3. AUTOREGRESSIVE REGRESSION MODEL FOR BLOOD LEAD ON HAND LEAD, FITTED IN LOG FORM, FOR BALTIMORE STUDY[a]**

| | Round | | | | | |
|---|---|---|---|---|---|---|
| Variable | 1 | 2 | 3 | 4 | 5 | 6 |
| Intercept ($\mu$g/dL) | 8.84[b] (0.45) | 2.50[b] (0.31) | 1.95[b] (0.45) | 1.12[b] (0.36) | 0.44 (0.27) | 0.85[b] (0.27) |
| Autoregression | NE | 0.662[b] (0.029 | 0.685[b] (0.048) | 0.693[b] (0.044) | 1.024[b] (0.042) | 0.884[b] (0.037) |
| Hand Lead ($\mu$g/pair $\times$ $10^3$) | 202.0[b] (41.1) | 13.7[b] (8.3) | 38.7 (25.8) | 61.7[b] (27.5) | -1.3 (12.7) | 29.8[b] (13.6) |
| N | 408 | 307 | 212 | 193 | 192 | 184 |
| Residual GSD | 1.666 | 1.323 | 1.380 | 1.336 | 1.256 | 1.235 |

Note: Soil abatement occurred between Rounds 3 and 4.
[a]NE = not estimated; values not in parentheses are model estimate parameter, standard error is shown in parentheses, GSD = geometric standard deviation.
[b]Statistically significant positive effect, one-tailed $p < 0.05$.

It appears then that children with higher exposures to interior dust or interior lead-based paint will more consistently have higher blood leads in these two neighborhoods than will children with exposure to exposure to elevated soil lead levels, and that it may be necessary to directly intervene to reduce dust and paint lead levels in these houses. Although exterior lead-based paint was correlated with soil lead, the addition of an exterior X-ray

TABLE 4-4. AUTOREGRESSIVE REGRESSION MODEL FOR BLOOD LEAD ON ENVIRONMENTAL LEAD, FITTED IN LOG FORM, FOR BALTIMORE STUDY, PREABATEMENT[a]

| Variable | Round 1 | Round 2 | Round 3 |
|---|---|---|---|
| Intercept | 7.77[b] | 0.69 | 1.38[b] |
| ($\mu$g/dL) | (1.19) | (0.72) | (0.53) |
| Autoregression | NE | 0.720[b] | 0.675[b] |
| | | (0.040) | (0.060) |
| Hand Lead | 45.8 | 0.0 | 67.8[b] |
| ($\mu$g/pair $\times 10^3$) | (53.9) | (NE) | (31.8) |
| Soil Lead | 1.48 | 1.84[b] | 0.0 |
| ($\mu$g/g $\times 10^3$) | (1.83) | (0.82) | (NE) |
| Dust Lead | 4.04[b] | 1.04 | 0.0 |
| ($\mu$g/m$^2$ $\times 10^3$) | (2.38) | (0.89) | (NE) |
| Exterior Paint | 0.066 | 0.0 | 0.0 |
| (mg/cm$^2$) | (0.068) | (NE) | (NE) |
| Interior Paint | 0.442 | 0.196 | 0.147 |
| (mg/cm$^2$) | (0.276) | (0.120) | (0.157) |

[a]NE = not estimated, values not in parentheses are the model estimate parameter, the standard error is shown in parentheses.
[b]One-tailed $p < 0.05$; statistically significant positive effect.

fluorescence term to the model did not significantly improved the ability to estimate blood lead levels. This does not imply that there is no benefit to abatement of soil or exterior lead-based paint, however, because there clearly was a relationship between postabatement soil lead levels and blood lead that may indicate that reducing soil lead has reduced the soil component of interior dust lead loading.

The model adopted for Baltimore is shown in Figure 4-1 and results are shown in Table 6. The BAL SP group needed to be split into separate components. The nonabated units in BAL SP (now denoted BAL P-2) were similar in response, and these differed from the abated units in BAL SP (still denoted BAL SP). We therefore show three groups in our analyses. The models included blood lead levels at Rounds 3 and 4, environmental lead levels at Round 1, and postabatement soil lead levels in group BAL SP after Round 4 (February 1990 and January 1991, respectively).

## TABLE 4-5. AUTOREGRESSIVE REGRESSION MODEL FOR BLOOD LEAD ON ENVIRONMENTAL LEAD, FITTED IN LOG FORM, FOR BALTIMORE STUDY, POSTABATEMENT[a]

| | Round | | | | | |
|---|---|---|---|---|---|---|
| Variable | 4C | 4S | 5C | 5S | 6C | 6S |
| Intercept | 1.18[b] | 0.40 | -0.49 | -0.05 | 0.34 | 0.66 |
| ($\mu$g/dL) | (0.50) | (0.83) | (0.38) | (0.56) | (0.44) | (0.39) |
| Autoregression | 0.646[b] | 0.693[b] | 1.063[b] | 1.071[b] | 0.912[b] | 0.822[b] |
| | (0.069 | (0.059) | (0.048) | (0.071) | (0.063) | (.046) |
| Hand Lead | 112.2[b] | 45.4 | 0.6 | 0. | 25.4 | 27.9 |
| ($\mu$g/pair $\times$ $10^3$) | (59.3) | (33.6) | (1.3) | (NE) | (20.4) | (18.3) |
| Soil Lead | 0. | 13.18[b] | 0.56 | 0. | 0. | 8.16[b] |
| ($\mu$g/g $\times$ $10^3$) | (NE) | (6.06 | (0.45) | (NE) | (NE) | (4.15) |
| Dust Lead | 0.88 | 2.55[b] | 0. | 1.11 | 0. | 0. |
| ($\mu$g/m$^2$ $\times$ $10^3$) | (1.26) | (1.33) | (NE) | (1.21) | (NE) | (NE) |
| Exterior Paint | 0. | 0. | 0. | 0.064 | 0. | 0. |
| (mg/cm$^2$) | (NE) | (NE) | (NE) | (0.060) | (NE) | (NE) |
| Interior Paint | 0.035 | 0. | 0. | 0. | 0.190 | 0.295[b] |
| (mg/cm$^2$) | (0.144) | (NE) | (NE) | (NE) | (0.124) | (.103) |
| N | 82 | 83 | 96 | 81 | 85 | 74 |
| Residual GSD | 1.308 | 1.318 | 1.191 | 1.265 | 1.277 | 1.167 |

[a]NE = not estimated, values not in parentheses are the model estimate parameter, the standard error is shown in parentheses, GSD = geometric standard deviation.
[b]One-tailed $p < 0.05$; statistically significant positive effect.

Floor lead concentrations and floor lead loadings were not as consistently associated with blood lead levels as in the Boston study, whether measured by AAS or XRF. We developed a model that used preabatement dust lead concentration as an indicator of lead indoor exposure. Interior and exterior XRF levels were as indicators of lead-based paint, for direct exposure and as source terms for soil and dust lead.

Unlike Boston, the abatement or nonabatement groups corresponded to spatially distinct neighborhoods that also differed in some important preabatement characteristics. We must therefore allow for the possibility that the primary environmental pathways, and the

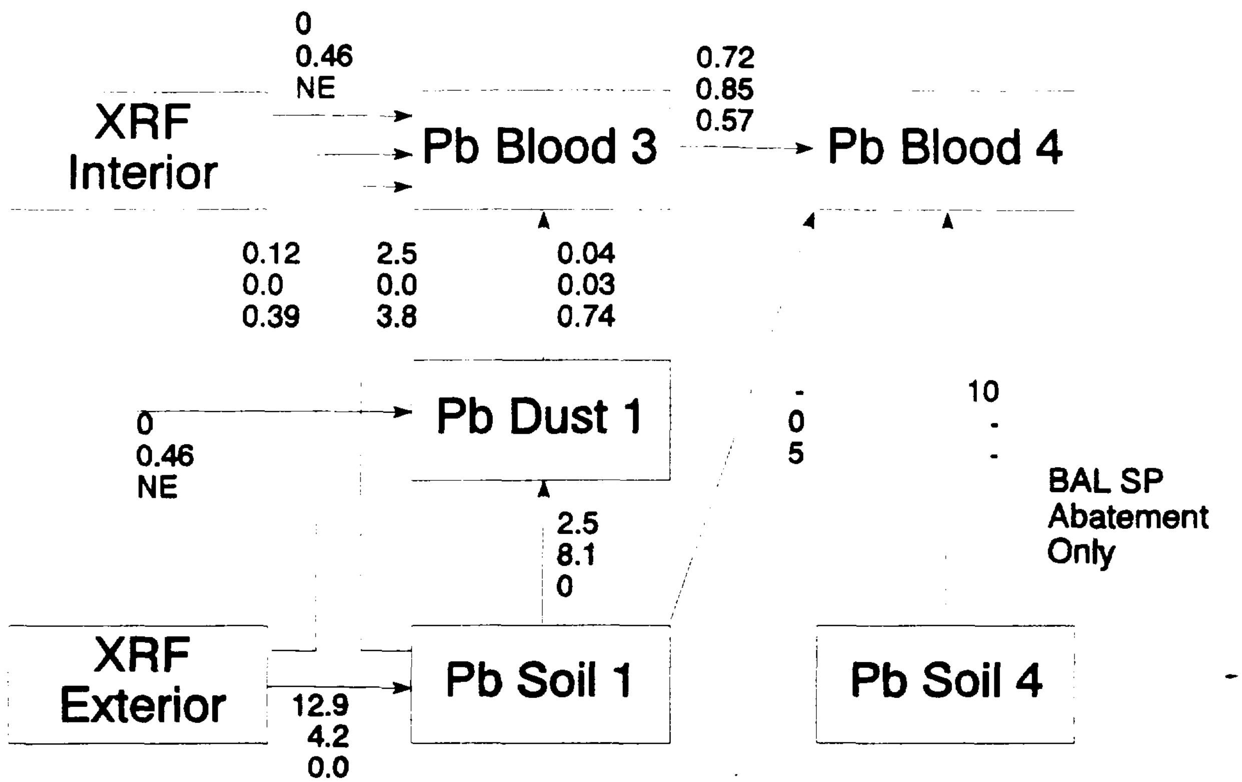


**Figure 4-1. Structural equation diagram for the Balitmore study. Numbers next to arrow, from Table 4-6, are the regression coefficients (top to bottom) for BAL SP, BAL P-1, and BAL P-2.**

effectiveness of the abatements in interdicting those pathways, may differ among different neighborhoods.

We found strong relationships between preabatement blood lead level and floor dust lead concentration in BAL P-2, and postabatement between blood lead level and soil lead concentration in BAL P-2. Exterior lead-based paint contributed to soil lead levels both preabatement in BAL SP (significant) and BAL P (positive but nonsignificant), but not in BAL P-2. Interior lead-based paint contributed significantly to floor dust lead preabatement in BAL SP, but not in BAL P.

There was at least some reasonable consistency in estimates of the blood lead autoregression coefficient R. The estimates decreased from 0.850 in BAL P to 0.720 in

**TABLE 4-6. REGRESSION COEFFICIENTS FOR BALTIMORE STRUCTURAL EQUATIONS MODEL, USING THE GLS METHOD**

| | | Response Variable (Output) | | | |
|---|---|---|---|---|---|
| | | Preabatement | | | Postabatement |
| Predictor Variable (Input) | Group | Blood | Dust Conc. | Soil Conc. | Blood |
| Preabatement Blood | BAL SP | | | | 0.72[a] |
| | BAL P-1 | | | | 0.85[a] |
| | BAL P-2 | | | | 0.571[a] |
| Preabatement Dust Conc. | BAL SP | 0.04 | | | 1 |
| | BAL P-1 | 0.03 | | | |
| | BAL P-2 | 0.74[a] | | | |
| Pre/Postabatement Soil Conc. | BAL SP | 2.53 | 2.50 | | 10.00[b] |
| | BAL P-1 | 0.0 | 8.11 | | 0.0 |
| | BAL P-2 | 3.79 | 0.0 | | 5.01[a] |
| Mean XRF Exterior Pb-Based Paint | BAL SP | 0.12 | | 12.86[c] | |
| | BAL P-1 | 0.0 | | 4.19 | |
| | BAL P-2 | 0.39 | | 0.0 | |
| Mean XRF Interior Pb-Based Paint | BAL SP | 0.0 | 628[c] | | |
| | BAL P-1 | 0.46 | 0.0 | | |
| | BAL P-2 | NE | NE | | |

[a]Significance level < 0.001.
[b]Postabatement soil lead used in this group.
[c]Significance level 0.01 to 0.05 one-tailed (0.02 to 0.10 two-tailed).

BAL SP, but to 0.571 in BAL P-2. The difference in R between BAL SP and BAL P-1 was only marginally significant in testing Hypothesis 1. (Hypothesis 1 asserts that R for nonabatement is greater than for effective abatement, so a one-tailed test is appropriate). In summary, *the relative decrease in blood lead levels between pre- and postabatement samples was not significantly larger in the soil abatement area BAL SP than in the designated nonabatement area BAL P, after adjusting for current exposure*. Effects of the abatements could only be detected after adjusting for changes in postabatement lead exposure.

### 4.3.2 Boston Study

The results for the Boston study are shown in Tables 4-7 and 4-8. The analysis of variance shown in Table 4-7 shows no significant difference among categories. However, there is a very significant difference among Rounds 1, 3, and 4 overall. The round by category interaction effects are statistically significant ($p = 0.0020$) as shown in Table 4-7.

**TABLE 4-7. REPEATED MEASURES ANALYSIS OF VARIANCE FOR LOG (BLOOD LEAD) FOR BOSTON STUDY, ANALYSIS OF VARIANCE TABLES**

| Effect | MS | df | p |
|---|---|---|---|
| | Between Subjects | | |
| Category | 0.0493 | 2 | 0.8729 |
| Variability | 0.3626 | 143 | |
| | Within Subjects | | |
| Round | 3.3324 | 2 | $< 10^{-6}$ |
| Round × Category | 0.2196 | 4 | 0.0020 |
| Variability | 0.0505 | 286 | |

The effectiveness of abatement is explored in more detail in the analysis of covariance in Table 4-8. Initial soil lead and dust lead levels in Table 4-8 show that the effects are not statistically significant. The lead paint variables showed even less predictiveness and are not shown here. The abatement interaction terms are about equally significant as shown in Table 4-8 ($p = 0.0024$). There is some marginal significance for the initial dust lead term ($p = 0.0787$) and for an interaction between initial soil lead and abatement effect as characterized by the round by soil lead interaction ($p = 0.0664$). Therefore, there is evidence for an effect of soil lead abatement on blood lead concentrations that depends—not surprisingly—on the initial soil and dust lead levels. Because soil lead and interior dust lead are highly correlated, we may infer that, in these Boston houses, exposure to dust lead (as a primary vector) that was derived from soil lead can be reduced over time by a combined removal of soil and dust lead, but not by dust lead abatement alone.

**TABLE 4-8. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR LOG (BLOOD LEAD) FOR BOSTON STUDY, ANALYSIS OF COVARIANCE TABLES**

| Effect | MS | df | p |
|---|---|---|---|
| Between Subjects | | | |
| Category | 0.1494 | 2 | 0.6442 |
| Log [PbS(1)] | 0.5899 | 1 | 0.1893 |
| Log [PbLDF(1)] | 1.0640 | 1 | 0.0787 |
| Variability | 0.3387 | 130 | |
| Within Subjects | | | |
| Round | 0.2255 | 2 | 0.0123 |
| Round × Category | 0.2144 | 4 | 0.0024 |
| Round × Log [PbS(1)] | 0.1381 | 2 | 0.0664 |
| Round × Log [PbLDF(1)] | 0.0510 | 2 | 0.3647 |
| Variability | 0.0504 | 260 | |

Additional analyses are shown in Table 4-9. An autoregressive regression model using hand lead as a surrogate exposure variable performs very well, with residual geometric standard deviations (GSD) of 1.33 to 1.38 that are nearly as small as any seen in regression models of cross-sectional studies. There is a large and highly significant autoregression coefficient for Round 4 blood leads on Round 3 blood leads taken 2 to 3 mo earlier (adjusted for hand lead) of 0.670, which captures the longitudinal course during the recontamination phase. This is very similar to the autoregression coefficient of 0.683 for postabatement blood lead at Round 3 on preabatement blood lead at Round 1.

Assessment of the predictiveness of other environmental lead variables is shown in Table 4-10. Hand lead is again predictive of blood lead, with coefficients that are not much different than those given in Table 4-9. Soil lead levels, including postabatement values, never made a significant contribution to increasing blood levels when hand lead and dust lead loadings were included in the model. Postabatement dust lead loadings were highly significant predictors of postabatement blood lead. There were significant reductions in blood lead between Rounds 1 and 3, with decreases of geometric mean blood lead relative to

## TABLE 4-9. AUTOREGRESSIVE REGRESSION MODEL FOR BLOOD LEAD ON HAND LEAD, FITTED IN LOG FORM, FOR BOSTON STUDY[a]

| Variable | Round 1 | Round 3 | Round 4 |
|---|---|---|---|
| Intercept | 9.74[b] | -0.89 | 4.26[b] |
| ($\mu$g/dL) | (0.73) | (0.72) | (0.61) |
| Autoregression | NE | 0.683[b] | 0.670[b] |
| | | (0.061) | (0.065) |
| Hand Lead | 148.8[b] | 107.4[b] | 1.56[b] |
| ($\mu$g/pair $\times$ $10^3$) | (49.3) | (35.0) | (18.4) |
| N | 150 | 146 | 143 |
| Residual GSD | 1.377 | 1.362 | 1.333 |

[a]NE = not estimated, values not in parentheses are the model estimate parameter, the standard error is shown in parentheses, GSD = geometric standard deviation.
[b]One-tailed $p < 0.05$; statistically significant positive effect.
Note: Abatement occurred between Rounds 1 and 3.

## TABLE 4-10. AUTOREGRESSIVE REGRESSION MODEL FOR BLOOD LEAD ON ENVIRONMENTAL LEAD, FITTED IN LOG FORM, FOR BOSTON STUDY[a]

| Variable | Round 1 | Round 3 | Round 4 |
|---|---|---|---|
| Intercept | 8.86[b] | 0.26 | 3.82[b] |
| ($\mu$g/dL) | (0.89) | (0.84) | (0.97) |
| Autoregression | NE | 0.666[b] | 0.646[b] |
| | | (0.062) | (0.082) |
| Hand Lead | 192.2[b] | 78.2[b] | 24.3 |
| ($\mu$g/pair $\times$ $10^3$) | (52.8) | (33.4) | (27.8) |
| Soil Lead | 0. | 0. | 0. |
| ($\mu$g/g $\times$ $10^3$) | (NE) | (NE) | (NE) |
| Dust Lead | 0.50 | 1.96[b] | 9.12[b] |
| ($\mu$g/m$^2$ $\times$ $10^3$) | (0.39) | (0.83) | (4.78) |
| Dust Abatement | -0.21 | -1.33 | -0.75 |
| Area Int. ($\mu$g/dL) | (0.39) | (0.53) | (0.68) |
| Soil Abatement | 0.74 | -0.70 | -0.75 |
| Area Int. ($\mu$g/dL) | (0.78) | (0.54) | (0.68) |
| N | 139 | 125 | 106 |
| Residual GSD | 1.364 | 1.343 | 1.329 |

[a]NE = not estimated, values not in parentheses are the model estimate parameter, the standard error is shown in parentheses, GSD = geometric standard deviation.
[b]One-tailed $p < 0.05$; statistically significant positive effect.
Note: Abatement occurred between Rounds 1 and 3.

control of 0.74 − (−0.70) = 1.44 μg/dL in the soil abatement group, and 0.74 − (−0.75) = 1.49 μg/dL between Rounds 1 and 4. For the group that received only dust abatement, there was an initial improvement in geometric mean blood lead between Rounds 1 and 3 of −0.21 − (−1.33) = 1.12 μg/dL, but after Round 3 there was a postabatement recontamination that increased blood lead to control levels. These differences are not the same as the raw blood lead mean differences because they have been adjusted for blood lead autoregression, hand lead, and dust lead.

The adopted model is shown in Figure 4-2. Regression coefficients are shown in Table 4-11. The model includes blood lead levels at Rounds 1 and 4 (October 1989 and September 1990, respectively). Dust lead levels at Rounds 1 and 4, and soil lead levels preabatement and postrecontamination were used. The mean XRF lead paint measurement was a better predictor than the maximum XRF or the total area of chipped and peeling paint, even though these were stabilized during the study. Analyses were run for each group BOS SPI, BOS PI, and BOS P.

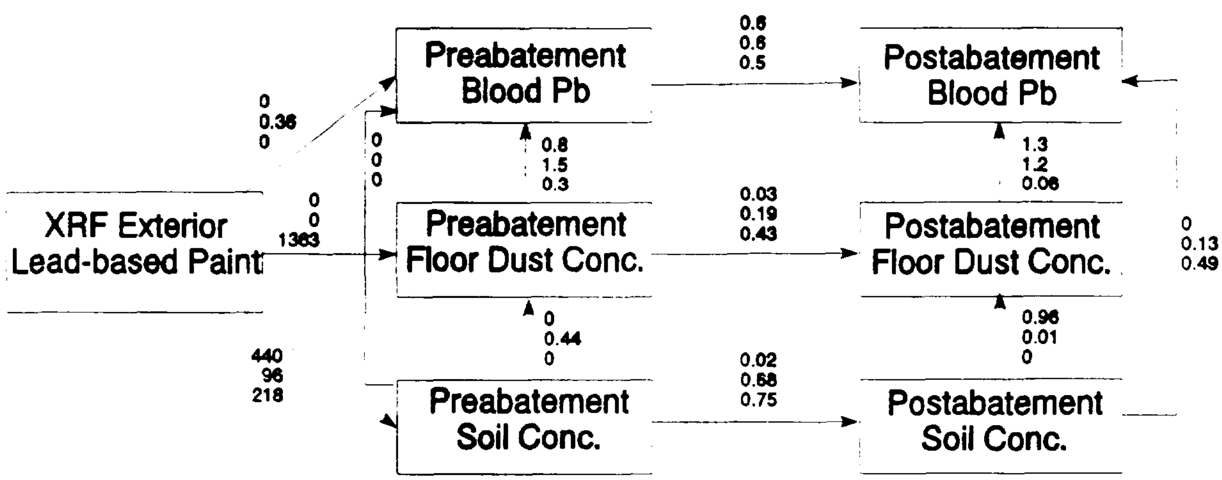


**Figure 4-2. Structural equation diagram for the Boston study. Numbers next to arrow, from Table 4-11, are the regression coefficients (top to bottom) for BOS SP, BOS PI, and BOS P.**

## TABLE 4-11. REGRESSION COEFFICIENTS FOR BOSTON STRUCTURAL EQUATIONS MODEL, USING THE AGLS METHOD

| | | Response Variable (Output) | | | | | |
|---|---|---|---|---|---|---|---|
| | | Preabatement | | | Postabatement | | |
| Predictor Variable (Input) | Group | Blood | Floor Dust Conc. | Soil | Blood | Floor Dust Conc. | Soil |
| Preabatement Blood Lead | BOS SPI | | | | 0.60[a] | | |
| | BOS PI | | | | 0.60[a] | | |
| | BOS P | | | | 0.51[b] | | |
| Preabatement Floor Dust Concentration | BOS SPI | 0.76[a] | | | | 0.03 | |
| | BOS PI | 1.45[e] | | | | 0.20 | |
| | BOS P | 0.25[c] | | | | 0.43[d] | |
| Postabatement Floor Dust Concentration | BOS SPI | | | | 1.33[e] | | |
| | BOS PI | | | | 1.18[a] | | |
| | BOS P | | | | 0.06 | | |
| Preabatement Soil Concentration | BOS SPI | 0.0 | 0.0 | | | 0.96 | 0.02 |
| | BOS PI | 0.0 | 0.44[e] | | | 0.01 | 0.68[a] |
| | BOS P | 0.0 | 0.0 | | | 0.0 | 0.75[a] |
| Postabatement Soil Concentration | BOS SPI | | | | 0.0 | 0.96[e] | |
| | BOS PI | | | | 0.13 | 0.011 | |
| | BOS P | | | | 0.49[e] | 0.0 | |
| Mean XRF Measure of Pb-Based Paint | BOS SPI | 0.0 | 0.0 | 440[a] | | | |
| | BOS PI | 0.36 | 0.0 | 96 | | | |
| | BOS P | 0.0 | 1,363[a] | 218[d] | | | |

[a]One-tailed p less than 0.0005.
[b]One-tailed p between 0.0005 and 0.005.
[c]One-tailed p between 0.005 and 0.025.
[d]One-tailed p between 0.025 and 0.05.
[e]One-tailed p between 0.05 and 0.10.

Preabatement blood lead was significantly related to preabatement dust lead, but not directly related to soil or paint lead. The indirect effects are very strong, however, from the paint → soil → dust → blood pathway. The component of soil lead not accounted for by lead-based paint also makes a large contribution to preabatement blood lead.

Postabatement blood lead levels are highly correlated with postabatement dust lead levels in the abatement groups BOS SPI and BOS PI, but not in the nonabatement group

BOS P. The coefficient (about 1.2 μg/dL blood lead per 1,000 ppm dust lead) is similar to that found in many other studies, and may be an underestimate since it does not represent blood lead that is fully equilibrated with the new dust lead levels. There is a small additional effect from postabatement soil lead that is not statistically significant.

Postabatement dust lead levels are significantly lower relative to preabatement dust lead concentrations in BOS PI than in BOS P, and much lower yet in BOS SPI. This is by far the most important contribution to reduced blood lead levels. Soil lead levels in the non-soil-abatement areas BOS PI and BOS P were also relatively lower after abatement, by about 25 to 30%.

A formal statistical test of the equality of the blood lead autoregression coefficient R was also carried out by constraining the coefficients to be equal in the three groups. There is no reason to believe that R is significantly different in the three groups.

In summary, *blood lead reductions in the Boston SPI group 8 mo after abatement appear to be associated with a persistent long-term reduction in the rate of transport of exterior lead (largely from soil) into household dust.* This amounted to about 1.2 μg Pb per 1,000 ppm dust lead reduction, or with a dust to soil lead ratio of about 0.7, about 0.8 to 0.9 μg/dL per 1,000 ppm soil lead. In the Boston PI group, a similar blood lead reduction could have occurred if there were no recontamination of household dust, but dust lead levels and blood lead levels did rebound to high preabatement levels. Stabilized lead-based paint did not contribute significantly to recontamination in the first few months after the abatement. Lead-based paint was associated with a significant part of the elevated lead levels in soil and dust found at the beginning of the study, contributing roughly 400 ppm to soil lead per mg $Pb/cm^2$ as a mean XRF level.

### 4.3.3 Cincinnati Study

The results for the Cincinnati study are shown in Tables 4-12 and 4-13. We found that the six neighborhoods in the study showed some puzzling differences, even within the same abatement category (Glencoe and Mohawk for controls and Findlay, Back, and Dandridge for dust abatement only in Year 1). We therefore carried all six neighborhoods in the analyses. The analysis of variance in Table 4-12 shows that there are significant differences among categories. However, there is a very significant difference among Rounds 1, 3, and

TABLE 4-12. REPEATED MEASURES ANALYSIS OF VARIANCE FOR LOG (BLOOD LEAD) FOR CINCINNATI STUDY, ANALYSIS OF VARIANCE TABLES

| Effect | MS | df | p |
|---|---|---|---|
| | Between Subjects | | |
| Category | 2.1850 | 5 | 0.0328 |
| Variability | 0.8752 | 169 | |
| | Within Subjects | | |
| Round | 1.9427 | 2 | $< 10^{-6}$ |
| Round × Category | 0.2790 | 10 | 0.0225 |
| Variability | 0.1316 | 338 | |

TABLE 4-13. REPEATED MEASURES ANALYSIS OF COVARIANCE FOR LOG (BLOOD LEAD) FOR CINCINNATI STUDY, ANALYSIS OF COVARIANCE TABLES

| Effect | MS | df | p |
|---|---|---|---|
| | Between Subjects | | |
| Category | 0.8589 | 5 | 0.2516 |
| Log (PbDF-R1) | 5.1040 | 1 | 0.0057 |
| Variability | 0.6457 | 135 | |
| | Within Subjects | | |
| Round | 0.3836 | 2 | 0.0265 |
| Round × Category | 0.3353 | 10 | 0.0006 |
| Round × Log (PbDF-R1) | 0.1903 | 2 | 0.1632 |
| Variability | 0.1043 | 270 | |

5 overall. The round by category interaction effects are statistically significant ($p = 0.0225$) as shown in Table 4-12. However, there is no obvious similarity in pattern between the two control neighborhoods. Two of the dust abatement neighborhoods (Findlay and Back) appear to have similar preabatement levels and response to dust abatement, but the Dandridge

neighborhood is different initially (Round 1) and very different postabatement (Round 5). Analyses were not extended to Round 9 due to unavailability of Round 9 environmental data. The initial Round 1 lead concentration in floor dust was the most predictive covariate and was used in the analysis of covariance in Table 4-13. Consideration of initial floor dust lead levels greatly increased the estimated effectiveness of the soil abatement between Rounds 1 and 5, amounting to a difference in interaction effects of $0.2132 - (-0.1233) = 0.3365$; that is, a reduction of $e^{0.3365} - 1 = 0.40$, or 40% reduction in blood lead, everything else being equal. However, this conclusion should not be over-interpreted because the baseline of the group average includes unexplained variation in the "control" groups. As has been often noted, the different Cincinnati neighborhoods appear to have been exposed before Round 5 to variable external lead sources that were not related to this study.

The autoregressive regression model results shown in Table 4-14 do not include hand lead levels, which were not available to us at the time of this analysis. It became evident that there were exposure-related differences among the groups that could not be adequately described by assigning different geometric mean blood lead levels to each neighborhood, except for Round 1. Thus, the first column in Table 4-14 shows blood lead levels or intercepts for a model that also includes dependence on floor dust lead concentration. The autoregressive models for Rounds 3 and 5 differ from the analyses in Tables 4-9 and 4-10, but are similar to the postabatement analyses in Tables 4-4 and 4-5, in that a separate autoregressive slope is used for each neighborhood. There are statistically significant differences in these coefficients even within abatement groups: between control neighborhoods Glencoe and Mohawk for Rounds 3 and 5 and between dust abatement neighborhoods Findlay and Dandridge at Round 3. Floor dust lead levels are highly predictive of within-group blood lead differences at Rounds 1 and 3, with a magnitude of about 2 $\mu$g/dL per milligram of lead per gram of dust, but not at Round 5. However, the residual GSDs are much larger than for the categorical repeated measures models in Tables 4-12 and 4-13, so this model is much less adequate than the autoregressive regression models for Baltimore and Boston.

The model adopted for Cincinnati is shown in Figure 4-3 and results are shown in Table 4-15. After careful evaluation of the preliminary results, it became evident that the CIN I-SE group needed to be split into separate components. The Back Street and Findlay

**TABLE 4-14. AUTOREGRESSIVE REGRESSION MODEL BY NEIGHBORHOOD FOR BLOOD LEAD ON ENVIRONMENTAL LEAD, FITTED IN LOG FORM, FOR CINCINNATI STUDY[a]**

| | Auto Regression | | | | | |
|---|---|---|---|---|---|---|
| Variable | Baseline | Round 3 | Round 5 | Round 5[b] | Round 7 | Round 9 |
| Intercept ($\mu$g/dL) | — | 2.37[c] (0.39) | 1.84[c] (0.58) | 1.46[c] (0.44) | 3.04[c] (0.24) | 2.43[c] (0.41) |
| Autoregression Round | — | 1 | 1 | 3 | 5 | 7 |
| Autoregression Glencoe (NT) | 7.64[c] (0.68) | 0.347[c] (0.058) | 0.517[c] (0.097) | 0.851[c] (0.177) | 0.496[c] (0.055) | 0.605[c] (0.082) |
| Autoregression Mohawk (NT) | 6.30[c] (1.20) | 0.510[c] (0.132) | 0.959[c] (0.252) | 1.484[c] (0.262) | 0.535[c] (0.056) | 0.897[c] (0.121) |
| Autoregression Findlay (I-SE) | 9.42[c] (1.09) | 0.546[c] (0.070) | 0.661[c] (0.105) | 0.870[c] (0.104) | 0.600[c] (0.050) | 0.820[c] (0.087) |
| Autoregression Back St. (I-SE) | 11.32[c] (1.67) | 0.598[c] (0.099) | 0.428[c] (0.151) | 0.567[c] (0.150) | 0.431[c] (0.098) | 0.586[c] (0.223) |
| Autoregression Dandridge (I-SE) | 11.15[c] (1.15) | 0.577[c] (0.067) | 0.673[c] (0.102) | 0.855[c] (0.095) | 0.618[c] (0.052) | 0.861[c] (0.083) |
| Autoregression Pendleton (SEI) | 8.66[c] (0.82) | 0.429[c] (0.063) | 0.667[c] (0.112) | 0.894[c] (0.104) | 0.638[c] (0.058) | 0.676[c] (0.078) |
| Hand Lead ($\mu$g/pair $\times 10^3$) | 53.0[c] (30.2) | 81.1[c] (53.3) | −12.6 (13.0) | −26.1 (10.7) | −2.6 (14.3) | 14.4 (10.8) |

[a]NE = not estimated, values not in parentheses are the model estimate parameter, the standard error is shown in parentheses.
[b]Alternate analyses.
[c]One-tailed $p < 0.05$; statistically significant positive effect.

neighborhoods (now denoted CIN I-SE1) were similar in response, and these differed substantially from the Dandridge neighborhood (now denoted CIN I-SE2). We therefore show four groups in our analyses. The models included blood lead and environmental lead levels at Rounds 1 and 5 (July 1989 and July 1990, respectively).

Floor lead concentrations and floor lead loadings were not as consistently associated with blood lead levels as in the Boston study. Dust lead concentrations or loadings at the entrance of the residence unit were better predictors than floor lead levels. We developed a

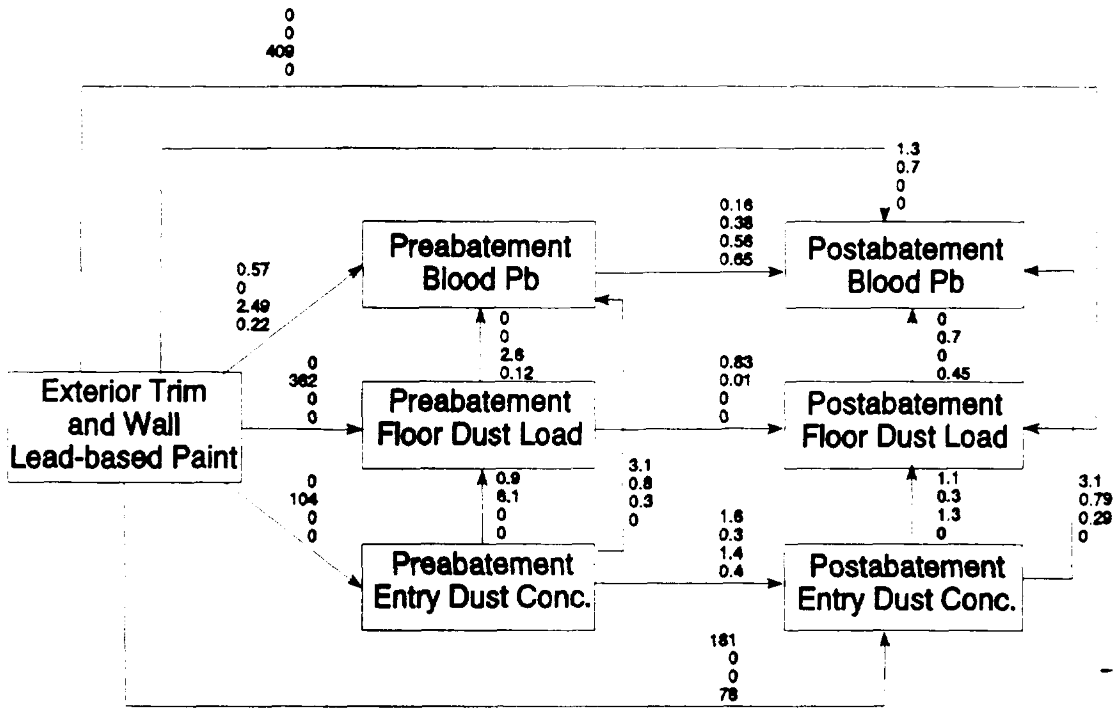


**Figure 4-3. Structural equation diagram for the Cincinnati study. Numbers next to arrow, from Table 4-15, are the regression coefficient (top to bottom) for CIN SEI, CIN I-SE-1, CIN I-SE-2, and CIN NT.**

model that used entrance dust lead concentration as an indicator of external lead exposure, and floor dust lead loading as a proximate indicator of indoor exposure.

Unlike Boston, the abatement groups corresponded to spatially distinct neighborhoods that also differed in some important preabatement characteristics. We must therefore allow for the possibility that the primary environmental pathways, and the effectiveness of the abatements in interdicting those pathways, do differ among different neighborhoods.

The lead-based paint measurements used in our models included interior wall and trim (denoted XRWL1 and XRTRM1 respectively) and exterior wall and trim (denoted XREWL and XRETRM, respectively). There were significant XRF levels ( greater than 1 mg Pb/cm$^2$) at least somewhere in many of these units, even the majority were described as rehabilitated ("gut rehab"). Only 7 of 157 children lived in nonrehabilitated units. The analyses were done with and without these children, all of whom lived in I-SE1.

## TABLE 4-15. REGRESSION COEFFICIENTS FOR CINCINNATI STRUCTURAL EQUATIONS MODEL, USING THE AGLS METHOD

| | | Response Variable (Output) | | | | | |
|---|---|---|---|---|---|---|---|
| | | Preabatement | | | Postabatement | | |
| Predictor Variable (Input) | Group | Blood | Floor Loading | Entry Conc. | Blood | Floor Loading | Entry Conc. |
| Preabatement Blood Lead | CIN SEI | | | | 0.162 | | |
| | CIN I-SE-1 | | | | 0.38[b] | | |
| | CIN I-SE-2 | | | | 0.564[a] | | |
| | CIN NT | | | | 0.649[a] | | |
| Preabatement Floor Dust Loading | CIN SEI | 0.0 | | | | 0.829[a] | |
| | CIN I-SE-1 | 0.0 | | | | 0.012 | |
| | CIN I-SE-2 | 2.61 | | | | 0.0 | |
| | CIN NT | 0.12 | | | | 0.0 | |
| Postabatement Floor Loading | CIN SEI | | | | 0.0 | | |
| | CIN I-SE-1 | | | | 0.69[e] | | |
| | CIN I-SE-2 | | | | 0.0 | | |
| | CIN NT | | | | 0.45 | | |
| Preabatement Entry Dust Conc. | CIN SEI | 3.13[e] | 0.94[d] | | | | 1.56[a] |
| | CIN I-SE-1 | 0.79 | 6.11[a] | | | | 0.30[d] |
| | CIN I-SE-2 | 0.29 | 0.0 | | | | 1.42[a] |
| | CIN NT | 0.0 | 0.0 | | | | 0.38 |
| Postabatement Entry Conc. | CIN SEI | | | | 1.12 | 0.0 | |
| | CIN I-SE-1 | | | | 0.28 | 0.048 | |
| | CIN I-SE-2 | | | | 1.32[d] | 0.035 | |
| | CIN NT | | | | 0.0 | 0.199 | |
| XRF Exterior Trim Pb-Based Paint | CIN SEI | 0.57 | 0.0 | 0.0 | 1.28[c] | 0.0 | 181[a] |
| | CIN I-SE-1 | 0.0 | 362[d] | 104[d] | 0.18 | 0.0 | 0.0 |
| | CIN I-SE-2 | 2.49[d] | 0.0 | 0.0 | 0.0 | 409[a] | 0.0 |
| | CIN NT | 0.22 | 0.0 | 0.0 | 0.0 | 0.0 | 78 |
| XRF Exterior Wall Pb-Based Paint | CIN SEI | 0.0 | 506 | 0.0 | 0.0 | | 230 |
| | CIN I-SE-1 | 1.38 | 1,810 | 774 | 1.24 | | 0.0 |
| | CIN I-SE-2 | 0.24 | 0.0 | 0.0 | 1.21 | | 260 |
| | CIN NT | 0.0 | 0.0 | 166 | 0.0 | | 131 |

[a]One-tailed p less than 0.0005.
[b]One-tailed p between 0.0005 and 0.005.
[c]One-tailed p between 0.005 and 0.025.
[d]One-tailed p between 0.025 and 0.05.
[e]One-tailed p between 0.05 and 0.10.

We found strong relationships between blood lead level and floor dust lead loading in CIN I-SE2 and in CIN SEI, both pre- and postabatement, and between blood lead level and entrance dust lead concentration postabatement in both groups, but only in CIN SEI preabatement. Exterior lead-based paint contributed to blood lead levels both pre- and postabatement in CIN I-SE1 and CIN I-SE2, but not in CIN SEI or CIN NT. Exterior lead-based paint contributed to floor dust lead loadings preabatement in CIN I-SE1 and CIN SEI, but not in CIN I-SE2 or CIN NT. Exterior lead-based paint contributed to entrance dust lead concentrations postabatement in CIN I-SE2, CIN NT, and CIN SEI, but not in CIN I-SE1. This may be accounted for to some extent because the autoregression of entrance lead postabatement to entrance lead concentration preabatement was 0.30 to 0.38 for CIN I-SE1 and CIN NT, but 1.42 to 1.56 for CIN I-SE2 and CIN SEI.

There was at least some reasonable consistency in estimates of the blood lead autoregression coefficient R. The estimates decreased from 0.649 in CIN NT to 0.564 in CIN I-SE2 to 0.380 in CIN I-SE1 to 0.162 in CIN SEI, as per Hypothesis 1. The difference between R for CIN NT and CIN SEI was statistically significant, $p = 0.0255$ one-tailed (Hypothesis 1 asserts that R for nonabatement is greater than for effective abatement, so a one-tailed test is appropriate). In summary, *in spite of the large changes in dust lead loadings and concentrations, increasing in some neighborhoods and decreasing in other neighborhoods, the relative decrease in blood lead levels between pre- and postabatement samples was significantly larger in the soil abatement area than in the nonabatement areas, after adjusting for current exposure.* The relative blood lead reduction was also larger in the areas with interior dust abatement only (albeit not significantly). It appears that, whatever transient effectiveness the soil and dust abatements may have had in the first few months after abatement, these effects were being overtaken by increases in lead exposure that presumably could not be controlled by the study. Effects of the abatements could only be detected after adjusting for changes in postabatement lead exposure.

## 4.4 DISCUSSION AND CONCLUSIONS

### 4.4.1 Comparison Across the Three Studies

The effectiveness of soil lead abatement in reducing blood lead varied greatly among the three cities. The variability in abatement effects is probably due to substantial differences in lead sources and pathways among the neighborhoods in these studies. We will discuss these differences for each study.

The Baltimore study had two neighborhoods, Upper Park Heights and Walbrook Junction. The area to which abatement was assigned (Park Heights) had enrolled families whose residences did not have soil lead levels that were high enough to justify abatement. The soil lead levels in the nonabatement premises in Park Heights that were measured in the preabatement phase were not significantly smaller than those of the abated houses or of the control premises in Walbrook Junction. We therefore used the nonabatement houses in Park Heights as an additional control group. Unlike the other studies, the soil abatement was not accompanied by interior dust abatement. There was essentially no significant effect of soil abatement in the abated houses, compared to the control group. Statistical covariate adjustment in both repeated measures analyses and autoregressive regression analyses showed that the differences in blood lead levels both before and after abatement were significantly dose-related to interior lead-based paint and (nonabated) interior dust. It is likely that interior paint contributed to child lead exposure, either directly by ingestion of paint chips, or indirectly by the hand-to-mouth exposure pathway:

$$\text{interior paint} \Rightarrow \text{interior dust} \Rightarrow \text{hands} \Rightarrow \text{blood.}$$

Cross-sectional and longitudinal structural equation analyses could be used to explore this hypothesis. However, because there were no repeated measurements of household dust lead, it will be very difficult to assess changes in exposure over time except by use of hand lead data.

*It is likely that soil lead abatement had little effect on the primary factors responsible for elevated child blood lead levels in these two neighborhoods, which appear to be interior lead-based paint and interior dust lead.*

The Boston study was conducted with blood and hand leads measured at one preabatement round and at postabatement rounds about 2 and 8 mo after abatement. Soil and dust lead measurements were available for all three rounds at about the same time. There were also environmental data on soil and dust immediately after abatement for the soil abatement premises, and there were dust lead measurements immediately after abatement for the dust-only abatement premises. These data allowed a very complete analysis of blood lead responses to changes in dust and soil lead over time. The results showed clearly that there was a persistent 10% reduction in blood lead levels (1.4 or 1.5 $\mu$g/dL) in the soil lead abatement children, and that, on average, the postabatement blood leads were lowest in premises that had the lowest postabatement soil lead and dust lead loadings. Interior and exterior lead paint were not significant predictors of blood lead for Boston children.

*When dust lead and soil lead levels show a persistent decrease as a result of effective abatement, blood lead levels also show a persistent decline. The postabatement blood lead levels are lower when postabatement dust lead levels are persistently lower over a long time.*

The Cincinnati study had collected blood and lead and environmental samples in six Cincinnati neighborhoods. We were able to generate analyses comparable to those reported for the Baltimore and Boston studies. After some analyses using models similar to those for Baltimore and Boston, it became evident that the neighborhoods within each treatment group (two neighborhoods as controls, three as interior dust abatement only) were not identical in some ways, so the analyses were run with six neighborhood-treatment groups instead of three. Although the statistical tests showed that there were strong interactions between group and round, there was no clear pattern of effect within any group except for a modest decrease in blood leads in the surface soil abatement neighborhood and in one of the dust abatement neighborhoods in Round 5. This reduction was hard to interpret because there were changes in blood lead in the control neighborhoods during this same time interval. Although there was a strong dependence of blood lead on environmental lead, particularly on hand lead and on current or previous dust lead loadings on floors, there was no clear pattern of change or response of interior dust lead levels after abatement.

Some of the analyses in Tables 4-14 and 4-16 require further discussion. The intercept corresponds roughly to the component of new lead exposure associated with "background" sources such as diet. We have assumed that the background sources were the same in all neighborhoods. The autoregression coefficient is a composite of time-dependent factors. If everything else were constant (stationary over time), then the autoregression coefficient would be equal to an exponential function of the pharmacokinetic mean residence time and the time between successive blood lead measurements. However, the relationship of blood lead measurements to a previously measured blood lead concentration is also a function of child age, seasonal variations, and other changes in lead exposure over time. We have not adjusted the autoregressions for these factors.

Note that the autoregression coefficient for Round 3 versus Round 1 blood lead is much lower in the soil abatement neighborhood (Pendleton) than in the three neighborhoods that received only interior dust lead abatement. For example, in Table 4-14, the difference between Dandridge (dust abatement only) and Pendleton (soil and dust abatement) for Round 3 versus Round 1 is $0.577 - 0.429 = 0.146$. In other words, the nonbackground (presumably soil and dust) steady-state component of blood lead was about 14% smaller in the soil abatement neighborhood than in a nearby neighborhood with dust abatement only. This suggests that there is at least some initial effect of soil lead abatement over and above that of dust lead abatement. The control neighborhoods, Glencoe and Mohawk, appear to have very different autocorrelation coefficients and other statistics. The control neighborhoods were at some distance from the soil abatement neighborhood, and the three indoor dust abatement neighborhoods were between them. The clustering of neighborhood locations and blood lead response variables suggests that some simpler comparisons may be needed in order to compare the effectiveness of soil lead treatment versus nontreatment.

The autocorrelations for the soil abatement and dust abatement neighborhoods were similar (differences were not statistically significant) after Round 3. This suggests that the environmental abatements did not affect hand and dust lead pathways, but may have reduced the overall soil and dust lead exposure. These preliminary findings and interpretations should be verified by more thorough longitudinal statistical analyses using structural equation models.

**TABLE 4-16. AUTOREGRESSIVE REGRESSION MODEL FOR BLOOD LEAD ON ENVIRONMENTAL LEAD, FITTED IN LOG FORM, FOR CINCINNATI STUDY[a]**

| Variable | Round 1 | Round 3 | Round 5 | Round 5[b] | Round 7 | Round 9 |
|---|---|---|---|---|---|---|
| Intercept ($\mu$g/dL) | — | | 3.46[c]<br>(0.64) | 2.54[c]<br>(0.55) | 3.64[c]<br>(0.61) | |
| Autoregression Round | Intercept | 1 | 1 | 3 | 5 | 7 |
| Autoregression Glencoe (NT) | 7.70[c]<br>(0.67) | 0.357[c]<br>(0.060) | 0.296[c]<br>(0.087) | 0.585[c]<br>(0.110) | 0.467[c]<br>(0.111) | |
| Autoregression Mohawk (NT) | 5.82[c]<br>(1.13) | 0.491[c]<br>(0) | 0.581[c]<br>(0.195) | 1.160[c]<br>(0.237) | 0.559[c]<br>(0.161) | |
| Autoregression Findlay (I-SE) | 9.40[c]<br>(1.06) | 0.511[c]<br>(0.071) | 0.467[c]<br>(0.106) | 0.709[c]<br>(0.105) | 0.557[c]<br>(0.097) | |
| Autoregression Back St. (I-SE) | 11.24[c]<br>(1.56) | 0.612[c]<br>(0.102) | 0.498[c]<br>(0.192) | 0.650[c]<br>(0.185) | 0.283[c]<br>(0.127) | |
| Autoregression Dandridge (I-SE) | 11.61[c]<br>(1.16) | 0.586[c]<br>(0.070) | 0.451[c]<br>(0.098) | 0.692[c]<br>(0.098) | 0.490[c]<br>(0.090) | |
| Autoregression Pendleton (SEI) | 9.06[c]<br>(0.88) | 0.415[c]<br>(0.067) | 0.400[c]<br>(0.106) | 0.716[c]<br>(0.120) | 0.663[c]<br>(0.122) | |
| Hand Lead ($\mu$g/pair $\times 10^3$) | 35.7[c]<br>(22.2) | 73.2<br>(56.8) | 64.1[c]<br>(33.9) | 34.2<br>(29.8) | 8.6<br>(38.5) | |
| Floor Dust Lead Loading ($\mu$g/m$^2 \times 10^3$) | 0.086<br>(0.111) | 0.089<br>(0.100) | 2.49<br>(1.97) | 0.38<br>(1.66) | 1.17<br>(1.91) | |
| Dust Round | 1 | 1 | 4 | 4 | 4 | |
| Floor Dust Lead Loading ($\mu$g/m$^2 \times 10^3$) | — | 4.58<br>(2.93) | —<br>(2.93) | —<br>(2.93) | 0.80[c]<br>(0.40) | |
| Dust Round | | 3 | | | 6 | |

[a]Values not in parentheses are the model estimate parameter, the standard error is shown in parentheses.
[b]Alternate analyses.
[c]One-tailed $p < 0.05$; significant positive effect.

There are some differences between Tables 4-14 and 4-16. The estimated regression coefficients between blood lead and hand lead for Rounds 3 through 9 are nonsignificant or negative in Table 4-14. When floor dust lead loadings were also included as predictors of blood lead, the estimated relation of hand lead to blood lead was always positive, although significant only for Round 5. When the hand lead regression coefficient was not significant, then the dust lead loading regression coefficient was significant or nearly so. This suggests that there are some interactions among blood lead, hand lead, and dust lead variables, possibly as causal pathways.

We are inclined to accept the conclusion of the Cincinnati investigators that blood and dust lead levels were affected differently at different times and places by exogenous sources, possibly related to repainting of nearby highway bridges or other events not under their control. However, the dose-dependence exhibited in the models suggests that reducing interior dust lead levels did reduce blood lead levels, at least for a while. The problem is that the abatements did not always persistently reduce dust lead levels.

*We conclude that there were additional sources of environmental lead exposure that had different effects on the neighborhoods during the course of the study and were not related to the abatement methods used in the Cincinnati study. It will be necessary to use other analysis methods, such as structural equations modeling, in order to assign changes in Cincinnati child blood lead levels to changes in lead exposure.*

## 4.5 SUMMARY OF STATISTICAL INFERENCES

This report concurs with the results reported by the individual studies. The reanalysis of the individual study data sets, where performed in the same manner as the report, revealed no evident errors in statistical analysis. Reconstruction of the exact statistical procedures and selection of records to be included proved to be a formidable task.

Certain procedures were not performed by one or more of the three studies, but were included in the reanalysis of the data. The repeated measures analysis and autoregressive regression model revealed that the relationship between environmental lead and blood lead was more or less uniform across all three studies. When the environmental lead increased, the blood lead increased, and when environmental lead decreased, blood lead decreased. The removal of variance caused by seasonal cycles and long-term time trends appeared to resolve most of the nonenvironmental variance in the blood lead measurements. The imputing of values for missing data in the Baltimore study restored enough observations to the data set to suggest a relationship between environmental lead and blood lead similar to that found in Boston, although the difference in Baltimore was not statistically significant.

Several of the statistical analyses confirm, or at least support, the observations in Chapter 3 that there is a strong link between environmental lead and blood lead. The most crucial analysis requires the development of structural equation models, a time-consuming process that will not be completed within the time limits of this report.

# 5. CONCLUSIONS

## 5.1 SUMMARY OF PROJECT

This project focuses on the exposure environment of the individual child. One measure of short-term exposure is the child's blood lead. Two other indicators of exposure are house dust lead concentrations and hand dust lead loading. From the perspective of the child's environment, changes in the soil lead concentration are expected to bring about changes in the house dust and blood lead concentrations, and hand dust loading. In each of the three studies, the soil lead concentrations were reduced to approximately 50 $\mu g/g$ in the study area. For most children, there was a measurable, although not always statistically significant, reduction of blood lead. When corrected for seasonal and age-related cyclic variations on blood lead, the impact was even greater, and the effect was maximized when the rate of movement of dust through the human environment was taken into account. That is, when street dust and house dust were also removed from the environment so that the clean soil represented the major source of lead to the child's environment, the impact of abatement was the greatest.

The earlier sections of this document evaluated the following statements:

(1) that the abatement of the soil resulted in a reduction of soil lead concentration on a case-by-case basis, and that this reduction persisted throughout the study (Chapter 3);

(2) that the intermediate exposure elements (street dust, house dust, and hand dust) responded to this soil abatement (Chapter 4); and

(3) that there was a measurable decrease in blood lead that could be directly related to the abatement of soil (Chapter 4).

Chapter 3 discussed the measurement and abatement of lead in soil. It was apparent that where soil abatement was conducted, this abatement was effective and persisted for the duration of the study. Chapter 4 provided the statistical analysis required to support the conclusions in Chapter 5.

In the Cincinnati study, the persistency of external dust abatement was very short, leading to the conclusion that sources of lead other than soil were predominant in the neighborhood. At this time, there is no further evidence for the nature or size of this source. It is clear, however, that until this source is identified and controlled, soil abatement at the neighborhood level under conditions similar to those in Cincinnati would have little influence on the house dust of individual homes and would only influence the blood lead concentrations for those children playing in the areas with the soil abatement.

## 5.2 SUMMARY OF RESULTS

The results of these three studies demonstrated a clear relationship between environmental lead and blood lead. There were few instances where changes in the blood lead concentrations could not be attributed to changes in environmental lead. Unexpected changes, such as the dramatic increase in the Cincinnati dust lead concentrations between November 1989 and July 1990, were observed in nearly every instance (floor, window, mat, and entry) of every study group, but the sources remain unexplained. Although this apparent contamination appears to have overwhelmed the intervention efforts, the fact that both hand dust loads and blood lead concentrations responded accordingly gives credence to the strong link between environmental lead and blood lead. Other, less dramatic changes also produced corresponding changes in blood lead concentrations.

In terms of changes attributed to intervention, it is appropriate to note that all three studies observed a quantifiable change in response to intervention. The analyses in Chapter 4 show that, although not always statistically significant, this quantifiable response to intervention is consistent even at low levels of environmental lead. Normalized to a decrease in soil lead concentration of 1,000 $\mu g/g$, the aggregate response in blood lead concentrations appears to be about 1 $\mu g/dL$. This suggests that there is no plateau, within the ranges measured in this project, where the removal of environmental lead will not produce a corresponding reduction in blood lead concentrations.

Finally, the project results shed additional light on the well-known phenomenon of seasonal cycles in blood lead concentrations. The rare opportunity to evaluate three independent longitudinal studies with similar sampling and analysis protocols led to the conclusion that the amplitude of the cycle is roughly 15% in all three cities, that the peak occurs about August 15-20, and that these factors appear to be independent of environmental lead.

## 5.3 SUMMARY OF STATISTICAL INFERENCES

### 5.3.1 Baltimore Study

It is likely that soil lead abatement had little effect on the primary factors responsible for elevated child blood lead levels in these two neighborhoods, which appear to be interior lead-based paint and interior dust lead.

It appears then that children with higher exposures to interior dust or interior lead-based paint will more consistently have higher blood lead concentrations in these two neighborhoods than will children with elevated soil lead levels, and that it may be necessary to directly intervene to reduce dust and paint lead levels in these houses. Although exterior lead-based paint was correlated with soil lead, the addition of an exterior paint term to the model did not significantly improve the ability to estimate blood lead levels. This does not imply that there is no benefit to abatement of soil or exterior lead-based paint, however, because there was clearly a relationship between postabatement soil lead levels and blood lead that may indicate that reducing soil lead has reduced the soil component of interior dust lead loading.

### 5.3.2 Boston Study

There is evidence for an effect of soil lead abatement on blood lead concentrations that depends—not surprisingly—on the initial soil and dust lead levels. Because soil lead and interior dust lead are highly correlated, we may infer that in the Boston houses studied, exposure to dust lead (as a primary vector) that was derived from soil lead can be reduced over time by a combined removal of soil and dust lead, but not by dust lead abatement alone.

Soil lead levels, including postabatement values, never made a significant contribution to increasing blood levels when hand lead and dust lead loadings were included in the model. Postabatement dust lead loadings were highly significant predictors of postabatement blood lead. There were significant reductions in blood lead between Rounds 1 and 3, with decreases of geometric mean blood lead relative to control of $0.74 - (-0.70) = 1.44$ $\mu$g/dL in the soil abatement group, and $0.74 - (-0.75) = 1.49$ $\mu$g/dL between Rounds 1 and 4. For the group that received only dust abatement, there was an initial improvement in geometric mean blood lead between Rounds 1 and 3 of $-0.21 - (-1.33) = 1.12$ $\mu$g/dL, but after Round 3 there was a postabatement recontamination that increased blood lead to control levels. These differences are not the same as the raw blood lead mean differences, because they have been adjusted for blood lead autoregression, hand lead, and dust lead. However, they point to a very clear conclusion from the Boston study: When dust lead and soil lead levels show a persistent decrease as a result of effective abatement, blood lead levels also show a persistent decline. The postabatement blood lead levels are lower when postabatement dust lead levels are persistently lower over a long time.

### 5.3.3 Cincinnati Study

We conclude that there were unexplained sources of environmental lead exposure that had different effects on the neighborhoods during the course of the study and were not related to the abatement methods used in the Cincinnati study. It will be necessary to use other analysis methods such as structural equations modeling in order to assign changes in Cincinnati child blood lead levels to changes in lead exposure. Lead paint was absent and was not a confounding factor.

## 5.4 INTEGRATED PROJECT CONCLUSIONS

This report concurs with the conclusions reported by the individual studies. The reanalysis of the individual study data sets, where performed in the same manner as the report, revealed no evident errors in statistical analysis. The repeated measures analysis and autoregressive regression model revealed that the relationship between environmental lead and blood lead was more or less uniform across all three studies. When the environmental lead increased, the blood lead increased, and when the environmental lead decreases, the blood lead decreases.

The removal of variance caused by seasonal cycles and long-term time trends appeared to resolve most of the nonenvironmental variance in the blood lead measurements. The imputing of values for missing data in the Baltimore study restored enough observations to the data set to suggest a relationship between environmental lead and blood lead similar to that found in Boston, although the difference in Baltimore was not statistically significant.

Several of the statistical analyses confirm, or at least support, the observations in Chapter 3 that there is a strong link between environmental lead and blood lead. The most crucial analysis requires the development of structural equation models, which were beyond the scope of this reanalysis.

### 5.4.1 Findings

The analysis of the data from the three studies showed evidence that blood lead responds to changes in environmental lead. This suggests that abatement of any type and to any degree will cause a reduction in the blood lead of children. All three studies and all groups within each study produced data supporting this conclusion, although not statistically significant in two of the groups in the Baltimore study.

All three studies also showed evidence for a quantifiable impact of intervention. This may have been intervention from soil abatement, dust abatement, or paint stabilization. In Baltimore, this impact was temporary at best and was marginally significant. In Cincinnati, the impact was quickly swamped by other sources of environmental lead. In Boston, the impact was persistent. The best estimate for this effect is 1 $\mu$g/dL per 1,000 $\mu$g/g decrease in soil. Similar decreases in exterior dust would be expected to have a similar effect.

There is evidence from all three studies that lead moves throughout the child's environment. This means that lead in soil becomes lead in street or playground dust, lead in paint becomes lead in soil, and lead in street dust becomes lead in house dust. A more detailed analysis of the data may show the relative contribution from two or more sources, but the present analyses confirm that this transfer takes place. In the Baltimore study, there was statistical evidence for implied causal pathways, such as paint to exterior dust, but in the Boston and Cincinnati studies, the pathways were explicit.

Finally, there is evidence for the continued impact of nonabated sources following abatement. This means that abatement of soil probably does not reduce the contribution of paint lead to the child's exposure.

### 5.4.2 Implications

In spite of the recent successes in reducing exposure to lead by removing lead from gasoline and canned food, lead exposure remains a complex issue. This integrated report attempts to assess exposure to lead in soil and house dust. It is only one component of the risk assessment process and cannot by itself be the sole basis for a risk management decision. However, the observations and conclusions are based on sound scientific measurements and reasonable interpretations of these measurements. A thorough understanding of the results of this project can provide guidance for regulatory decisions and public health policies.

This report concludes that a reduction in environmental lead corresponding to a decrease of 1,000 $\mu g/g$ in soil will result in a reduction of about 1 $\mu g/dL$ in blood lead. Although this modest decrease suggests that soil abatement as a form of environmental intervention would not be particularly effective in clinical treatment of a lead poisoned child, in an environmental intervention program where the goal is to reduce the incidence of blood lead concentrations above 10 $\mu g/dL$, this small change could reduce this incidence by 10 to 15%.

Lead in soil and lead-based paint are closely linked in the child's environment. If there is exterior lead-based paint, then soil lead is likely to be elevated. If there is interior lead-based paint, then measures to reduce the impact of soil lead on house dust will be ineffective. Public health programs designed to reduce lead exposure will not achieve that objective unless both paint and soil abatement are implemented.

From a regulatory standpoint, where the goal is to determine a safe level of lead in soil, this report concludes that abatement of soil above 500 $\mu g/g$ will measurably reduce blood lead concentrations. It does not say that this reduction in blood lead would be permanent or cost-effective.

From another perspective, decisions about soil abatement are likely to be made on an individual home basis or on a neighborhood basis. For an individual home, the owner or renter may require only peace of mind in knowing that the property is safe for children if the soil lead concentrations are below an acceptable level, or, if not, that soil abatement would be a cost effective way to reduce or eliminate the problem.

This project shows that, on an individual house basis, soil abatement reduces the flow of lead into the home and its incorporation into house dust. The magnitude of this reduction depends on the concentration of lead in the soil, the amount of soil-derived dust that moves into the home, and the frequency of cleaning in the home. The number and ages of children and the presence of indoor/outdoor pets are factors known to increase this rate, whereas the frequency of cleaning with an effective vacuum cleaner and removing shoes at the door serve to reduce the impact of soil lead on house dust.

For multifamily homes, the study shows that the location of the living unit may also be a factor. Lead from soil reaches the second and third floor of an apartment building.

On a neighborhood basis, the focus of concern is usually directed at the local public health officer, who faces a risk management decision for which an exposure assessment based on the results of this project is only one element. Guidance in this case should provide general exposure scenario information that would assist the officer in predicting blood lead concentrations should soil be abated.

In summary, there are many options, other than soil abatement, available to the individual home owner or renter, that are not practical for the public health officer making a decision on the neighborhood level.

### 5.4.3 Recommendations

This project demonstrated that abatement of soil can, in some urban environments, reduce children's blood lead, and that this reduction can be quantified with sophisticated statistical techniques. On matters of public health concern, this is an important conclusion that will perhaps lend guidance to decision makers in the immediate future. In the long term, however, the information gathered here has provided long overdue explanations of some of the processes that influence childhood lead exposure. Yet there is much more to be learned. The following suggestions may give some direction to the further analysis of these data and the planning of similar studies.

1. Continued reanalysis of the data by independent investigators will reveal even more information about the movement of lead in urban environments. These analyses should include more structural equation modeling, meta analysis, and GIS analysis.

2. New techniques to measure dust in urban neighborhoods should be developed. The mat placement experiment in the Cincinnati study shows promise of being a simpler approach to measuring house dust than other methods available. If developed further to determine the optimum time of placement and recovery, a public health officer could place these mats and recover them with minimum intrusion into the home and reasonable convenience in recovering the sample in the laboratory.

   Other techniques, such as hand wipe analysis and methods for measuring dust loading as well as dust lead concentration should be further developed and become routine is studies of this type.

3. Judgements on whether or not to conduct soil abatement should be based on more than the expected decrease in blood lead concentrations. This study shows that children living in urban environments are exposed to many sources of lead. Abatement of one source should be considered only in the context of other sources, including lead-based paint. This decision should also take into the consideration the impact on children moving into the neighborhood from areas with lower exposure. Effective abatement prior to this move would probably have a greater impact on these children than on those residing in the neighborhood before abatement.

# 6. REFERENCES

Annest, J.L.; Pirkle, J.L.; Makuc, D.; Neese, J.W.; Bayse, D.D.; Kovar, M.G. (1983) Chronological trend in blood lead levels between 1976 and 1980. N. Engl. J. Med. 308:1373-1377.

Barnett and Lewis (1984) Outliers in Statistical Data. John Wiley and Sons, NY.

Bentler, P.M. (1989) EQS Structural Equations Program Manual. BMDP Statistical Software, Los Angeles, CA.

Dempster, A.P.; Laird, N.M.; Rubin, D.B. (1977) Maximum likelihood from incomplete data via the EM algorithm. J. Royal Statistical Soc. Series B, Vol 38: 1-11.

Grant, L.D.; Elias, R.W.; Goyer, R.; Nicholson, W.; Olem, H. (1990) Indirect health effects associated with acidic deposition. National Acid Precipitation Assessment Program. SOS/T Report 23. U.S. Government Printing Office, Washington, DC.

Hasselblad, V.; Stead, A.G.; Galke, W. (1980) Analysis of coarsely grouped data from the lognormal distribution. J. Am. Stat. Assoc. 75:771-778.

Lotus Development Corporation (1990) Lotus 1-2-3 Release 3.1. Lotus Development Corporation, Cambridge, MA.

Orchard, T.; Woodbury, M.A. (1972) A missing information principle: Theory and applications. Proc 6th Berkeley Symp on Mathematical Statistics and Probability. Vol 1, pp 697-715.

Roberts, J.W.; Camaan, D.E.; Spittler, T.M. (1991) Reducing lead exposure from remodeling and soil track-in in older homes. Air and Waste Management Association Paper 91-134.2, 84th Annual Meeting and Exhibition, Vancouver, British Columbia, June 16-21, 1991.

U.S. Environmental Protection Agency (1986) Air quality criteria for lead. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no EPA-600/8-83/028aF-dF. 4v. Available from: NTIS, Springfield, VA; PB87-142378.

Wilkinson, L. (1990) SYSTAT: The system for statistics. Version 5.03 (1991). Evanston, IL: SYSTAT, Inc.

Woodbury, M.; Hasselblad, V. (1970) Maximum likelihood estimates of the variance covariance matrix from the multivariate normal. Proc of SHARE XXXIV, Vol 3, pp 1550-1561.



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